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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely, will make the Journal a fund of extensive information readily available to students, teachers, researchers, and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain therefore those papers of scientific and practical interest which are presented at the annual meetings of that society in the January issues including 1933. This issue of the Journal contains the papers read at the nineteenth annual meeting held in Buffalo, New York, September 8-9-10, 1932. The July, 1933 number will contain the abstracts of articles on periodontia which appeared in the years 1931 and 1932, complete.

Announcements

THE CHICAGO CENTENNIAL DENTAL CONGRESS

AUG. 7TH-12TH, 1933

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THE AMERICAN ACADEMY OF PERIODONTOLOGY

Twentieth Annual Meeting August 3, 4, 5, 1933

HOTEL STEVENS, CHICAGO.

Recent Findings In Dental Pathology and Diagnosis[1]

By RUDOLF KRONFELD, M. D.,
Chicago, Ill.

THE rapid progress of the biological sciences within the past decade has contributed largely to the solution of many problems of dental pathology. In the field of periodontal diseases, this progress is expressed most vividly in the changing type of specimens used for the description of both normal and pathologic tissue conditions in the teeth and surrounding structures. A survey of the bibliography reveals the fact that most scientists depended upon specimens of animal teeth for their studies. Many of the leading investigators in this country used specimens of sheep's teeth; other authors reproduced specimens of dogs' or cats' teeth. But there is nothing said as to the applicability of the findings in these animals to conditions in human structures. It is, in fact, very doubtful whether one is justified in using the findings to draw the conclusions that have previously been drawn from them.

From the foregoing the need for proper human specimens becomes evident. After all, we are dealing with human patients; and with full recognition of the value of investigations in sheep or cat, the final word will be spoken by the findings in human specimens. Fortunately, during the last few years an increasing number of dental laboratory workers have taken up the systematic microscopic study of specimens of human jaw tissues, for which it is indispensable that human jaws of individuals of different ages be procured. The specimens must be removed from the body immediately following death, must be properly fixed, and then must be decalcified and embedded in celloidin. Sections must be prepared that show the teeth in their true relationship to the surrounding hard and soft tissues without tears or other preparatory artifacts. In specimens of this type normal and pathologic conditions are found in the ratio in which they occur in the mouth of the average individual. Only from such specimens can conclusions be drawn as to diagnosis and therapy in man.

In the study of human specimens, the radiographic examination of the tissues and the comparison of the radiographic findings with the actual tissue changes as revealed by the microscope is of great importance. If human jaws that have been removed during autopsy are examined radiographically, they will always reveal a variety of conditions similar to those found in the mouths of patients. However, while in the case of patients, our diagnosis is based largely upon experience and analogy and only rarely upon biopsy, in the case of jaw specimens, the areas under consideration can be sectioned and examined microscopically, and the radiograph can be compared with the actual underlying tissue conditions.

Despite extensive controversy and a large number of publications on the

[1]Read before the American Academy of Periodontology, Buffalo, N. Y., September 10, 1932.
From the Research Department, Chicago College of Dental Surgery, Dental Department, Loyola University.

subject, there is still no uniformity of opinion as to what constitutes a normal gingival crevice. However, these discussions have brought about an increasing interest in the study of the histology of human gingival tissues, and these studies have, in turn, yielded some very interesting results. One of these results is the revised conception concerning the depth of the gingival crevice. In the first place, all the old statements that a normal gingival crevice is one, two, three, or any set number of millimeters deep are incorrect. The depth of the human gingival crevice is subject to wide individual variations. Of these variations the most important one is the gingival crevice of zero depth. Gottlieb has for the first time called attention to the fact that a gingival crevice may have no depth at all, and he considers such crevices ideal.

I shall demonstrate a number of such crevices without depth from patients of different ages and from different teeth (Fig. 1). All these specimens have one thing in common, namely, that the intact gingival epithelium is attached to the tooth surface without the presence of a crevice. The location of these extremely shallow crevices on the tooth surface is subject to variations. In young individuals, the gingival crevice is located on the enamel. With advancing age it approaches the cemento-enamel junction until, in older individuals, the bottom of the gingival crevice is found on the surface of the cementum. During this entire process, clinically known as recession or senile atrophy of the gingivae, the crevice may remain extremely shallow. This observation is of greatest importance from a practical viewpoint. Crevices with zero depth and intact epithelial covering are observed, as a rule, only in clean, well-kept mouths. The better the oral hygiene, the shallower are the crevices. The crevice with zero depth is the ideal since it is the cleanest form of crevice that one can imagine and offers the least possibility for the development of pathologic conditions.

As stated before, the depth of the gingival crevice depends largely upon oral hygiene and cleanliness. In young teeth that have just erupted into the oral cavity, the crevices, as a rule, are very shallow, usually having no depth at all. Unless good care is taken of the gingival tissues by proper brushing and removal of calculus and other irritating factors, the crevices soon become deeper. The mechanism of this deepening is that the epithelial attachment continues to separate from the tooth surface while the irritated gingival tissues do not concurrently shrink and retract; thus a pocket of increasing depth develops between gingival margin and tooth surface. If, on the other hand, the mouth is kept scrupulously clean, the gradual separation of the epithelial attachment from the tooth surface will be followed by a corresponding shrinkage and retraction (atrophy) of the gingival margin, and the crevice will not become deeper.

In summarizing, it may be said that in the question of the gingival crevice, two concurrent factors are of primary importance: (1) the gradual detachment of the soft tissues from the tooth surface, and (2) the concurrent involution and atrophy of these detached tissues. The first of these two processes, the separation

of the soft tissues from the tooth surface, takes place throughout life; it is a physiologic phenomenon, a part of the regressive processes that occur in every living organism. The second process, the involution of the gingival tissues, depends largely upon the state of health of the oral cavity. If the gingival tissues are clean and healthy, their atrophy keeps pace with their detachment from the root surface, and the gingival crevice remains very shallow throughout life.

Extremely shallow crevices are not found, however, only in *healthy* conditions. Frequently the gingival margin is found completely destroyed under calcu-

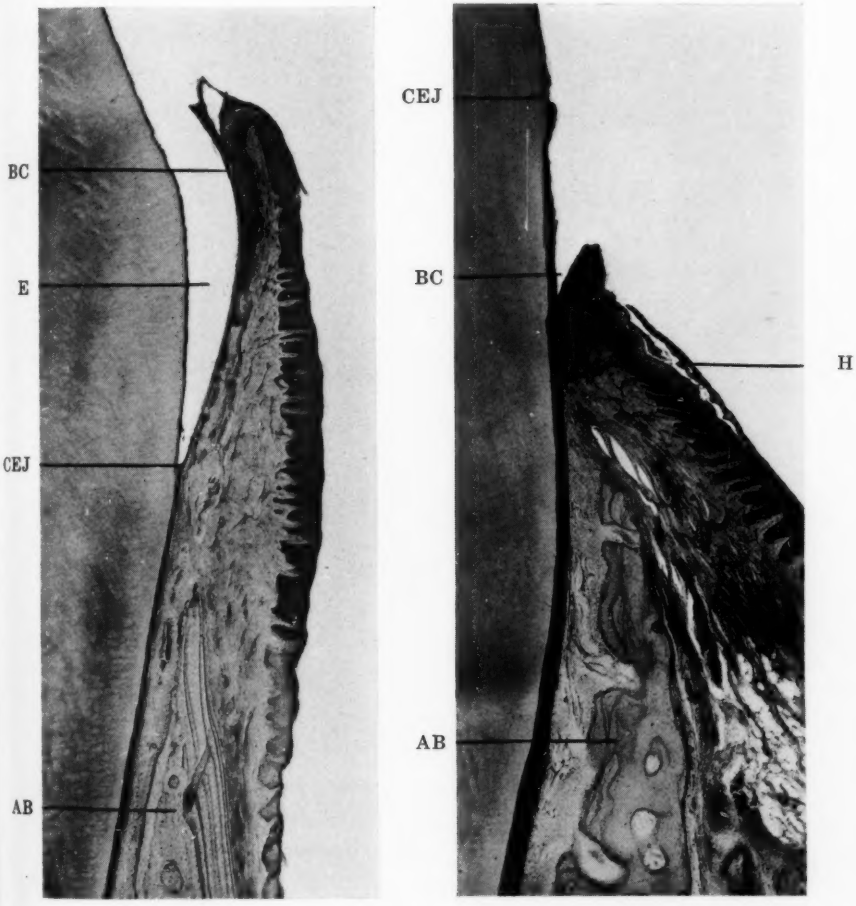

FIG. 1. Human gingival crevices of zero depth.

A—Lingual surface of upper cuspid. Age, fifteen years. Crevice of zero depth located on the surface of the enamel. E, enamel; CEJ, cemento-enamel junction; BC, bottom of gingival crevice; AB, alveolar bone.

B—Lingual surface of upper central incisor. Age, fifty-three years. Crevice of zero depth located on the surface of the cementum. CEJ, cemento-enamel junction; BC, bottom of gingival crevice; AB, alveolar bone; H, hornification of the gingiva.

lus, and the result is a flat, ulcerated gingival crest without any noticeable crevice. This condition can be illustrated in sections through human teeth with deposits of calculus (Fig. 2). Clinically these cases are characterized by very rapid healing and epithelization of the ulcerated surface following removal of the irritating deposits.

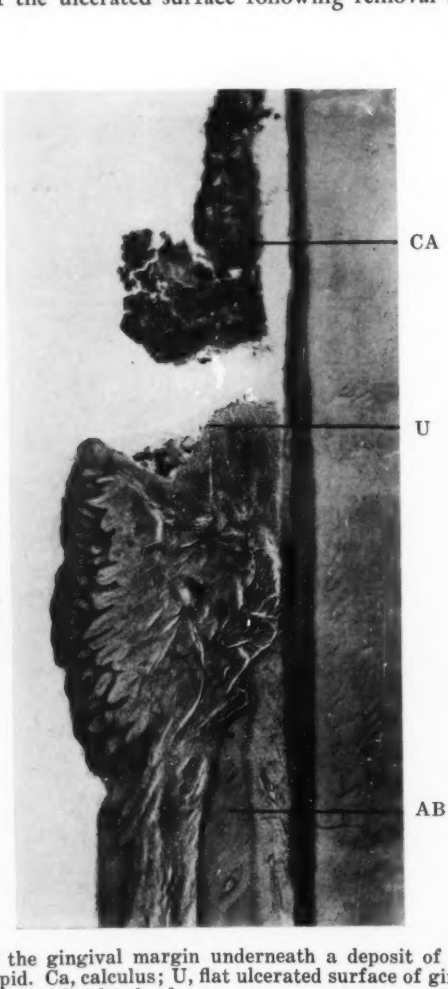

FIG. 2. Ulceration of the gingival margin underneath a deposit of calculus. Lingual surface of lower bicuspid. Ca, calculus; U, flat ulcerated surface of gingiva which was in contact with the calculus; AB, alveolar bone.

As has been indicated, very valuable information has been gained from a comparison of radiographic and microscopic findings in the same specimen. Fig. 3 illustrates a case that shows clearly the value as well as the limitations of radiographic diagnosis. The specimen under consideration is a lower second bicuspid which was in the mouth of a patient who had lost all molars by early extractions. No restoration had been made to replace the lost teeth. In the upper jaw all teeth

were present. As a result of the closing of the bite in combination with the loss of the first bicuspid, the second biscuspid was tipped forward, until at the time of the death of the individual, the long axis of the tooth had been tipped about 30° from the normal vertical position. The radiograph shows extensive loss of the alveolar bone except in a small area at the apex. The microscope reveals even a more extensive absence of bone than could be seen in the radiograph: the radiographic appearance of bone at the apex apparently is the result of an optical projection of the bone on the buccal and lingual side of the mandible over the apex; whereas, in a central section through the tooth, there is no alveolar bone whatsoever visible around the apex.

FIG. 3. Mesial tipping of a lower right second bicuspid after the loss of the neighboring teeth. Extensive destruction of the alveolar bone. The radiograph of the specimen in the upper left corner shows the amount of bone destruction. In a mesio-distal section through the jaw, the bicuspid appears embedded in fibrous connective tissue. Notice the location of the bottom of the gingival crevice on the root surface of the bicuspid. Despite the extensive loss of alveolar bone, about two-thirds of the surface of the anatomical root are still in organic connection with the surrounding tissues. The periodontal tissues of the lower cuspid are intact. (From Kronfeld, Histopathology of the Teeth and their Surrounding Structures, Lea & Febiger, Philadelphia).

The bicuspid is embedded in fibrous connective tissue that is attached to the apical two-thirds of the root surface, a fact which is of greatest practical significance. It illustrates very plainly that there is by no means a definite relationship between location of the bottom of the pocket and position of the alveolar crest. The bone has been resorbed as a result of occlusal pressure; however, the detachment of the remaining periodontal soft tissues did not follow the recession of the bone. The importance of this observation hardly can be over-emphasized as it shows that the usual procedure of diagnosing "pockets" from a radiograph is incorrect. The depth of a pocket is determined only by the line of attachment of the soft tissue; the amount of bone present is not a criterion. A radiograph can demonstrate

a pocket, therefore, only after a radiopaque material (for instance, a guttapercha point) has been introduced into the pocket and photographed along with the tooth. The only thing that can be seen in the routine radiograph is the amount of bone present; the supposition that loss of bone and formation of a pocket, namely, detachment of soft tissues, must necessarily correspond is not substantiated by the actual examination of the tissues involved.

In a discussion of the clinical prognosis of a tooth that is loosened by excessive occlusal stress, the attachment of the soft tissues again will have to be considered first and the condition of the bone second. For example, in the radiograph, the tooth illustrated here certainly does not look very promising. However, if the pockets were explored, it would be found that there is a considerable amount of soft tissue attachment still present; only about one-third of the anatomical root is exposed above the bottom of the crevice. Therefore, there is a distinct possibility that alveolar bone may regenerate around the lower two-thirds of the root, providing that the injurious stress is relieved. If, on the other hand, exploration of the pockets reveals that the detachment of the soft tissues has kept pace with the bone destruction so that the greatest portion of the root surface is denuded, the prognosis would be hopeless; reattachment can hardly be expected under such circumstances, and without connective tissue attachment, bone regeneration is impossible.

The last part of my paper deals with the influence of increased and decreased functional stress upon the supporting tissues of human teeth. It is reasonable to assume that in an intact set of thirty-two teeth, without malocclusion and without interference of any kind, every tooth is subject to the proper and adequate amount of functional stress. Such a set of teeth in perfect condition and alignment is, however, extremely rare among our civilized races. Usually some teeth are destroyed or lost by caries; improper occlusal conditions are created by malocclusions and by dental restorations, and as a result, teeth are subject to an abnormally increased or decreased amount of functional stress. The question arises: how do the supporting tissues react to additional or diminished functional demands? The answer to this question can be given best by a functional analysis of human jaw specimens. In almost all jaw specimens some teeth are missing; as a result the antagonistic teeth are subject to a distinct decrease in functional stress. Other teeth take over part of the chewing activities of the missing teeth. They can, therefore, be considered as teeth with increased function. A comparison of the microscopic appearance of periodontal membrane and alveolar bone of these two groups of teeth gives valuable information on tissue changes under increased and decreased function.

The periodontal membrance of a tooth exposed to normal occlusal stress is characterized by the presence of strong, well-developed fibre bundles. Especially the oblique fibres show a very dense, regular arrangement running parallel to each other and leaving only narrow spaces for the vessels and nerves between them. In a functionless tooth the fibres are so scarce that hardly any sharply defined

bundles can be found; only a few strands are present that run in different directions through the periodontal space, sometimes parallel to the root surface (Fig. 4).

In impacted teeth the periodontal membrane is sometimes replaced by fat tissue. The same change is found occasionally also in erupted teeth that have had no antagonists for a long time. Fig. 5 shows an area on the root surface of an

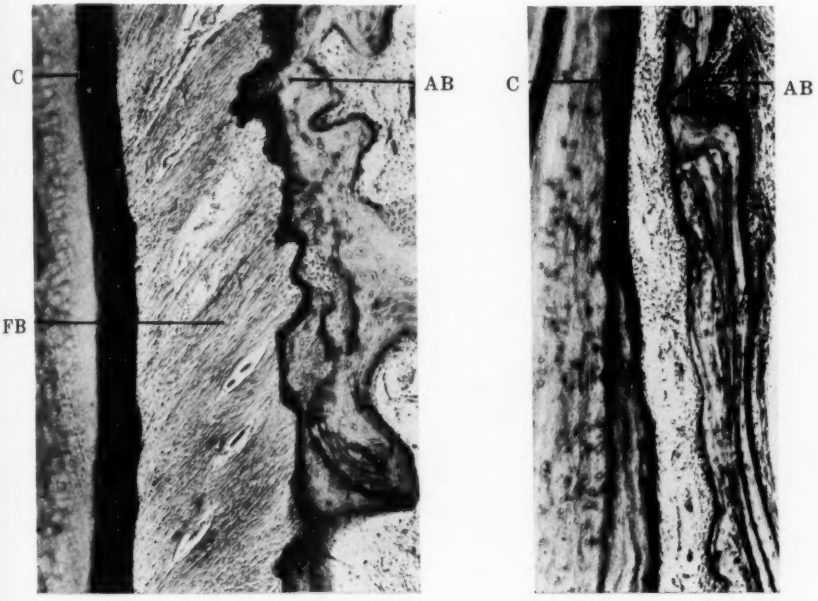

FIG. 4. Human periodontal membrane under different functional conditions.

A—Upper bicuspid in heavy end-to-end occlusion. Well-developed, strong fibre bundles of the periodontal membrane. C, cementum; FB, oblique fibre bundles of periodontal membrane; AB, alveolar bone.

B—Upper molar of the same jaw from which Fig. 4A was taken. Tooth without antagonist. Atrophic, thin periodontal membrane. C, cementum; AB, alveolar bone.

Note the difference in the width of the periodontal membrane in A and B: the periodontal membrane of the tooth in occlusion (A) is about three times as thick as that of the tooth without antagonist (B).

upper second molar from an old man whose mandible had been edentulous for several decades. In the lower part of the area reproduced here, there is still fibrous periodontal tissue present; the arrangement of the fibres is identical to that seen in Fig. 4; the periodontal space is very narrow. At FT, the space between bone and cementum is occupied by fat tissue; the distance between bone and root is wide and irregular. Evidently, in this part the periodontal membrane has lost entirely its biological and structural qualities; there is no definite relationship between tooth and bone, but the tooth surface simply has been included in the system of bone and marrow spaces, and the former "alveolar bone" runs through the fat marrow in the same manner as the trabeculae of cancellous bone in the jaw.

Since all functional stimuli exerted upon a tooth are transmitted to the alveolar bone, the latter reacts promptly to the occlusal conditions present about the tooth. In order to understand these bone changes, it is necessary to say a few words about the normal physiology of the alveolar bone.

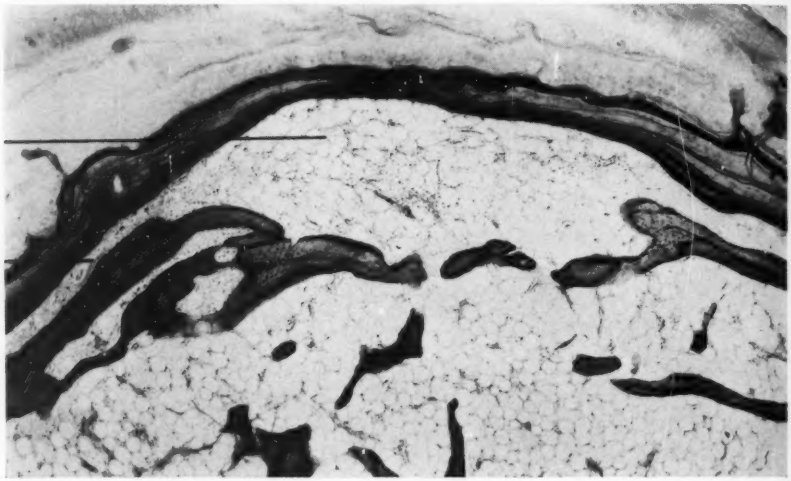

FIG. 5. Root surface of an upper molar that had been without antagonist for a long time. Part of the periodontal membrane has been replaced by fat marrow. PM, thin atrophic periodontal membrane, similar to that shown in Figure 4 B; FT, fat marrow in direct contact with the surface of the root.

The alveolar bone is normally subject to a constant process of building-up and tearing-down (formation and resorption). Resorption is stimulated by the presence of old portions of the bone that have reached their limit of life. This process of tissue aging is called senescense; it is found, with few exceptions, all over the organism (elimination of old cells and replacement by new cells). The eliminated portions of bone are normally replaced by newly built bone, and it must be emphasized that this new formation is largely controlled by functional factors. Under constant functional conditions, the total mass of bone will remain unchanged. The amount of bone that is resorbed is immediately replaced by new bone, the formation of which is necessitated by the prevailing functional demand; thus, tearing down and building up are balanced.

This balance can be disturbed in two ways, either by an increase in function or by a decrease in function. Increased function means increased mechanical stimulus and increased activity of the osteogenic cells; consequently, the physiologic resorptive loss will be overcompensated by excess new formation. We might say that for each resorbed unit of bone two new units are rebuilt; the result will be an increase in the total mass of bone. This increase is called functional hypertrophy. Functional hypertrophy is frequently observed in the bones under certain

functional conditions. A typical example is the increase in weight and thickness of the bones, induced by continued heavy physical labor.

If, on the other hand, function is decreased, the resorption will still go on as usual; but the resorbed portions of bone are no longer replaced by an equal amount of new bone because adequate functional demand is lacking. In this case we might say that for each two units of bone that are resorbed only one unit is rebuilt. This of course, leads to a reduction in the total mass of bone and is called bone atrophy (disuse atrophy, atrophy due to inactivity). This form of atrophy is found, for example, in the bones of the extremities if a limb is paralyzed or otherwise put out of function for a long time; resorption continues but there is no functional stimulus to cause new formation of bone, and the result is disuse atrophy of the bone.

In studying functional changes in the alveolar bone, it is necessary to distinguish between "alveolar" bone and "supporting" bone. The "alveolar" bone consists of the outer and the inner plate of the alveolus. The space between these two plates is occupied by the "supporting" bone, which consists of a network of trabecules connecting the plates of "alveolar" bone. It has been found that under changing functional conditions the "alveolar" bone remains rather constant; whereas, the "supporting" bone changes considerably. The greater the functional stress, the stronger and denser will be the "supporting" bone. In case of lack of function, the "supporting" bone will be almost entirely missing; only the thin outer and inner alveolar plates will be left.

In a broad sense, the entire mandible and maxilla belong to the "supporting" bone. Both jaws will undergo a considerable amount of change if certain functional conditions are present over a long period of time.

The difference in the structure and density of the alveolar bone around teeth under different functional conditions can be visualized and studied by comparing radiographs of such teeth. Where a number of teeth have been lost by extractions, a radiograph of the jaws will sometimes reveal a thickening and condensation of bone around the teeth that are still in occlusion (functional hypertrophy of bone). The bone will appear much thinner around the teeth that have lost their antagonists; the structure of the supporting bone will be hardly visible sometimes. Where all teeth are present and in normal occlusion, usually the same density of bone is found around all the teeth present.

In animal teeth it is possible to create experimentally certain abnormal functional conditions. This can be done either by decreasing or by increasing the normal functional stress. By grinding or eliminating one of a pair of antagonistic teeth, the function can be decreased; by cementing to teeth crowns or caps that interfere with normal occlusion, the occlusal stress can be increased.

The largest experiment of this type was made by Gottlieb, Orban and Kronfeld. The final results were published by Gottlieb and Orban in the form of a monograph. The technique was as follows: Metal caps were cemented upon upper

and lower right first molars of dogs. In some cases the caps had oblique occlusal planes, thus causing a horizontal (lateral) component of force and resulting in a tipping movement of the teeth. Other caps had horizontal occlusal planes, thus creating vertical force and causing depression of the teeth into their sockets. In the majority of cases, due to the excessive force applied, traumatic injuries to the periodontal tissues were produced.

If great stress is applied over a long period of time, the supporting bone and, finally, the configuration of the entire jaw is changed. One of the dogs used in the experiments of Gottlieb, Orban, and Kronfeld wore caps on the right upper and lower first molars for a period of thirteen months. After this time the animal was killed and the right and left molars sectioned in buccolingual direction. A comparison of the bone of the right and left side revealed a greater number and increased thickness of the trabecules of the supporting maxillary bone on the side on which the cap had been worn. A similar change took place in the mandible of a young dog who wore metal caps on the right first molars over a period of two months and ten days. The lower right first molar was depressed (intruded) by the heavy occlusal force. As a result the architecture of the entire mandible was changed, so that the body of the mandible on the right side is considerably lower and thicker than on the control side. This change in the configuration is the result of a gradual process of rearrangement and reconstruction of the mandible. Because of the increased occlusal pressure the bone at the bottom of the alveolus was resorbed, thus causing the tooth to be lowered; at the same time compensating deposition of new bone took place on the outer surface of the mandible, and thus the bone adapted itself to the changed functional conditions.

These findings in animal experiments together with our knowledge of analogous changes in human teeth contribute to a rather definite picture of the changes under various functional conditions. Before eruption, the periodontal membrane is thin and has no distinctly arranged fibre bundles. The surrounding bone does not show the dense arrangement of "supporting" bone. This condition may persist for a life-time if the tooth fails to erupt but remains impacted in the jaw. As the tooth erupts and reaches its normal occlusion, the periodontal space becomes wider; well-defined fibre bundles develop between root and bone; the "supporting" bone becomes reinforced so as to withstand functional stresses. If the functional demands are increased during life, the strengthening of the periodontal membrane and the reinforcement of the bone will be still more marked.

If, on the other hand, a formerly functioning tooth is put out of occlusion, either by destruction of its crown or by loss of the antagonistic teeth, the reverse process takes place. The periodontal membrane gradually loses its strong, fibrous structure, and the bone becomes atrophic.

* * *

Discussion

ARTHUR H. MERRITT, D. D. S., (New York City): Dr. Kronfeld called attention to the deep pocket and the two methods by which it might be treated.

If reattachment is not likely to take place, I would like to know whether by the conservative method of treatment that tooth could be put in health, provided that the pocket did not change markedly in its depth, as a result of the subgingival curettage and would the inner wall so heal over as to make it a safe risk for the patient?

If it does not, are we justified in leaving pockets of that depth which appear on the surface to be healthy? In other words, have we got finally to come back to the question of either reducing these pockets or subject our patients to a risk which, if we knew all it included, we might not be willing to take?

DR. KRONFELD: From the microscopic aspect of this pocket, it does not seem very promising or very probable that the lower part, the distinct pouch with a narrow entrance would heal or would ever be anything else but an area of chronic inflammation. We know from examples in general surgery that wherever a pouch has been established with a narrow entrance and wider bottom, with no access for cleaning, there is always a possibility of recurrence of inflammation. I do not doubt that by dilating the pocket and packing you may get an improvement, perhaps a complete epithelialization, and then there is certainly no objection against the retention of the flap overlying the pocket. But on the other hand I believe there will be always the possibility of a plugging up of the drainage and perhaps a sudden flare-up of a lateral abscess.

Question: I should like to ask, does the cementum increase in thickness with functional activity?

DR. KRONFELD: I will say we have never been able to establish a distinct connection between functional activity and thickness of cementum, but from all we have found, it points toward the fact that in functioning teeth the cementum is thinner than in functionless. We haven't very much exact data on this. The only way you can investigate the thickness of cementum is by comparing thickness on one side of the jaw with thickness on the other side of the jaw in the same mouth if one side is subject to function and on the other side the tooth has no antagonist. If your patient dies, then you can measure the thickness of cementum. In doing so we have found that usually the teeth that are out of function have thicker cementum. There seems to be a tendency toward extrusion out of the socket and as a result of this more cementum is deposited. But the question is extremely difficult to answer. In reality we know very little about the thickness of cementum.

CELIA RICH, D. D. S., (Nashville, Tenn.): In one of the first slides shown the X-ray findings were quite different from the histological findings; there was a great deal of soft tissue attachment altho the bone had been almost entirely destroyed. I would like to ask Dr. Kronfeld what would be the effect on the regenerative possibilities of that case if the operator severely traumatized those soft tissues.

DR. KRONFELD: The question Dr. Rich brought up is what I was aiming at when I discussed the slide. Some operators in a case like that, merely basing their treatment upon the X-ray diagnosis, are bound to do harm. Why? Because every tenth of a millimeter of soft tissue attachment that is left on the surface of a root is of value and may be the source of regeneration of bone. If the operator removes the soft tissue that surrounds this root, he deprives himself of the possibility of bone regeneration in the area that is denuded.

I believe that the right thing to do would be to take an explorer and if you slide down on the left surface of the tooth you will find there is very little pocket present. In other words, the soft tissue is attached right at the surface of the gum. I think there is no indication for surgery in such a case as this. By surgery all one does is to remove the bed for the regeneration of bone when there is no pocket present.

MEMBER: In many cases that are shown in which there is an apparent breaking-down, they are ascribed to traumatic occlusion. Is it really over-function or atrophy that is responsible? In other words, is it possible to produce a force upon a root that will increase the strength and density of the supporting tissues, but if increased beyond a certain point be heavy enough to break them down?

DR. KRONFELD: There is certainly such a thing as over-working a tooth and it is just a matter of clinical distinction. I always used to say, when the question came up, if you have a man in charge and want to make an athlete out of him, you make him do his physical work every day. If you compel him to do too much, it may be necessary for him to be carried to the hospital, but if you do it in a reasonable way, and watch his body reaction, you may make an athlete out of him; on the other hand, you may make a cripple out of him.

May I ask permission to say another word about reattachment?

I said that I was rather skeptical about it. On the other hand Dr. Coolidge, who is present here, has done some very interesting work along the line of repair following root canal therapy, has shown that in infected root ends there was regeneration of tissues around them following infection and filling of root canals or surgical removal of the apices, and there was also a distinct redeposition of new cementum and regeneration of fibers around these root ends; so I am contradicting myself. I said I did not believe there is a reattachment possible to infected cementum surface. On the other hand, in our own institutions we have found cases in which such a reattachment has occurred at the apex after treatment of root canals. But to my knowledge such redeposition of cementum and attachment of fibers as has been shown around the apices has as yet not been demonstrated in the field of a periodontal pocket, and there is a difference.

Question: Would you consider a non-cellular cementum as being capable of becoming highly infected?

DR. KRONFELD: We know that in a primary cementum, (cementum that does not contain cells), the fibers radiate and the radiation of fibers means the presence of fine channels. Also, though these are microscopic, they still are able to harbor bacteria because the space that is occupied by one single fiber forms a tunnel that can contain millions of bacteria. I think there is no doubt that even a thin layer of cementum can be infected by bacteria.

But there is a question whether the cementum is still a living surface or an inert, dead surface. You wouldn't expect an attachment to the enamel of an erupted tooth and the cementum of an erupted tooth is not very different, but shortly after extraction it is another story. The cementum still retains a certain amount of vitality and there is a reattachment possible because we know that we can replant teeth. You also know you can take a piece of bone and transfer it from one part of the body to another and it will heal in, but once the piece of bone has become a dead, inert sequestrum and once the cementum has become dead, we cannot expect a similar reaction.

The Prevention Of Periodontitis[1]

HAROLD KEITH BOX, D. D. S., PH. D., F. R. M. S.
Toronto, Canada

UNDER the heading of so-called pyorrhea in humans, two pure types of periodontal disease involving pocket-development and pus-formation are commonly presented in practice. Recognition of clinical entities with distinct grouping of symptoms, pathologic findings, and treatment, is obviously of great importance.

While within its strict meaning, the term pyorrhea means pus-flow, it is nevertheless now popularly accepted to indicate gingival suppuration, and it is hardly necessary to point out that its usage in dental nomenclature is of little value unless properly oriented to its different etiologic and pathologic backgrounds.

In the first type, pocket-formation is an early symptom and precedes tooth-mobility, with gingival inflammation a predominating feature, and deposits as a rule abundant. The pocket is usually wide, shallow and fairly slow in development with a predilection for the interproximal tissues, an exception being in cases of mouth-breathing. This disease I have designated Simplex Periodontitis, (1), which corresponds with the "Schmutz-pyorrhoe" of Gottlieb.

In my second type, termed Complex Periodontitis, the predominating symptom is not to be found in the gingival tissues. Pocket-formation has been preceded by profound structural changes in the deeper supporting tissues which have introduced an early mobility. The pocket is usually narrow, deep and fairly rapid in development. It occurs as a rule on the line of function, and on the side of tension or pull, and the base varies in position as related to the pocket opening.

Complex Periodontitis is generally localized in the periodontal tissues of teeth in traumatogenic occlusion. Gottlieb describes a somewhat similar type and designates it "Diffuse Alveolar Atrophy with Paradental Pyorrhea." Primary migration and loosening are characteristic early clinical symptoms, the pockets and suppuration occurring later.

The evidence in favour of the existence of fundamental lesions apart from gingival infection and its sequelae is so overwhelming that it is impossible to understand the viewpoint of those observers who consider the effects of gingival infection sufficient to explain all the clinical phenomena seen in the human periodontium.

Generally speaking, pure simplex cases are noted where occlusal conditions are good and oral hygiene is poor. Conversely, pure complex cases are noted where hygienic conditions are good and the occlusal conditions unfavorable. Fusions of both types are of course commonly found. Where occlusal conditions are good and oral hygienic conditions poor, and periodontitis is absent, it is apparent that factors local and general, are at work which control gingival infection. The

[1]Read before the American Academy of Periodontology, Buffalo, N. Y., September 10, 1932.

inhabitants of Tristan da Cunha largely have end-to-end bites, a favourable occlusal factor and use no artificial methods of oral hygiene like tooth-brushing, yet in the presence of a "certain amount of tartar," periodontitis is rare.

The prevalence of periodontal disease in animals is not generally appreciated by the dental profession. The noteworthy contributions of Colyer (2) have undoubtedly shed much light on this phase of the problem. Its widespread existence is indicated in his classification viz:

(a) wild animals in the captive state

(b) domestic animals

(c) wild animals in the natural state.

A discussion of these findings is beyond the scope of this paper, but it is significant to note that Colyer regards periodontal disease in animals as manifestations of Simplex Periodontitis. He has stated in his conclusions: "Expressed in biological language, the disease is due to an alteration in the environment of the teeth."

In a discussion of periodontal disease, as a whole, in order to understand my concept, it will be necessary to discuss briefly the following two subjects as they relate to the periodontal tissues, viz: certain basic states and infection.

1. Basic States—(a) local—functional adaptations

(b) general—temporary (influenza, pneumonia, etc.) prolonged (diabetes, nephritis, continued dietetic deficiency)

2. Infection

The local basic state in any given periodontium has been established by adaptation, or tendency to adaptation, of the supporting tissues to changed physical functional conditions. In a recent paper (3) read before the American Society of Orthodontists I called attention to some of the changes that occur in the pericementum following modifications of its physical function and pointed out the importance of an appreciation of these alterations in that broader analysis of aggregate changes where an attempt is made to determine the influence on this tissue of disturbed systemic states, nutritional and otherwise.

The following arbitrary division of occlusal function was made:

(1) Heavy function.

(2) Traumatogenic function.

(3) Functionless.

1. Heavy function falls into two classes, viz: increased stress within the limits of the reserve force of the cell, and overstress. It must be borne in mind that this is a most important distinction and one too frequently overlooked. Adami and McCrae (4) have stated: "If cells are put upon a contant not excessive stretch or even are intermittently exposed to such a stress, if they be well nourished, proliferation ensues, the distension acting as a stimulant." And again "mechanical

force exerted either as pressure or traction upon a cell is able to cause the growth and proliferation of that cell so long as at the same time there is no interference with its nutrition and the force acting on the cell is not excessive."

Increased stress inducing physiologic hypertrophy is found clinically in well balanced occlusions and articulations, and on certain teeth in increased axial loading following extensive tooth extraction, or where used for abutments, or in malocclusions.

The histologic picture of the pericementum shows hypertrophy of the fibre-bundles with decrease in the amount of indifferent supporting tissue.

Adami has pointed out that overstress or diminished nutrition will lead to the opposite condition of atrophy, in other words, atrophy is produced by excessive function in the presence of normal nutrition. He has stated: "Overwork carried beyond a given limit tends to produce cell exhaustion, if continued, to produce cell atrophy."

The point where the increased stress passes beyond the limits of the reserve force of the cell has not been determined. As the pericemental structure is the expression of the chemical constitution of the cells, and all histologic alterations presented may be regarded as expressions of adaptations to changed conditions, it follows that in the study of physical functional modifications, the answer is to be found in the tissues themselves.

2. Traumatogenic function. Trauma means injury produced mechanically and where found in the pericementum is the result of traumatogenic stress. Traumatogenic stress is an element in traumatogenic occlusion, and is also produced in certain types of orthodontic procedures.

Traumatic states commonly noted in the microscope include the following:

(a) Cementum fracture.

(b) Necrosis.

(c) Fibrosis. { non-inflammatory. / inflammatory—pericementitis fibrosa.

(d) Granulation tissue.

It is inexpedient in a paper of this length to discuss in detail the various histologic findings mentioned. It is interesting to note, however, that in the push zones where the marrow-spaces have been opened up, and are fused with, or are undergoing incorporation in the membrane, the marrow-tissue frequently shows a definite fibrosis. This tissue assumes a new function when merged with the pericementum and associated with this is shown a new morphology assuming the qualities of the membrane. That this is not a granulation tissue is shown by the absence of exudate cells, and an atypical vascularity; also the order of fibrosis is not like that following the healing of granulation tissue. Where as in pericementitis fibrosa, there is a slow but definite increase in fibrous tissue, it is to be noted that the constituent structures in the area are involved, principally the vascular

channels in which the arterioles are more affected than the venules. It is obvious that these changes interfere with the nutrition of bone and membrane and disturb the process of repair.

3. Functionless teeth. According to Adami, "the factors which are most potent in determining the state of a cell are nutrition and performance of function." He has stated that "if nutrition be good and there be loss of function, atrophy will supervene from disuse."

It has been shown by Kronfeld (5) that the pericementum of non-functioning teeth consists largely of a very loose tissue. This is in decided contrast to the membrane of teeth in heavy function within the limits of the reserve force of the cell where the fibre-bundles are well-developed and the zones of supporting tissue are small. Compared to this ideal normal, in the membrane of functionless teeth, the tissues lose their cellularity and striking architecture of cells and processes, with decrease in the number of fibres and density of fibrillar arrangement. We have given to these atrophic changes, the term, adaptive fibropenia.

Histologic findings of the pericementum are numerous where from a quantitative standpoint fibre-bundles and zones of fibropenia are found in many different ratios. I advanced the hypothesis that these findings, hitherto unexplained, in a great many instances may be looked upon as authentic records of the adaptation of the membrane to varied and fine modifications of physical function.

In the ideal normal of heavy function inducing hypertrophy of fibre-bundles and good development of supporting bone, the gingival tissues clinically as a rule exhibit a marked resistance to infection and in fact are best able to resist harmful influences of various kinds which tend to bring about morbid changes in them.

The local basic state has been established by adaptation to its multiple environment. This environment may be altered by local or general changes. That general systemic disturbances are frequently manifested in the oral cavity is common knowledge. In the presence of a good local basic state, it has been my experience that general systemic factors establish themselves with difficulty. Perfect functional conditions are rare, as abnormal occlusions are the rule. Clinical observation would indicate that manifestations of general systemic disease usually appear in direct ratio to the character of the local basic state, and in many cases with resulting rapid extension of established lesions. The observations of Cloutier (6) of the effects of influenza in this regard are in harmony with my own. When the dentist has established almost ideal functional conditions, and satisfactory results in the tissues are not achieved, a disturbed general basic state should be suspected, and the attention of the physician should be drawn to his failure so that he may attempt to correct the systemic factors. Our contact with patients frequently shows evidence of these general basic states in the oral cavity when the rest of the body is apparently adapting itself fairly well to them.

Periodontal infection is a process consisting in a growth of bacteria in the tissues, in the diffusion of their products, and in the reaction brought about by

their presence. Infection is part of our environment to which we have adapted ourselves both locally and generally. In the periodontium, as elsewhere, it localizes where the tissues are below par, that is to say they have used up or have not developed a reserve. Infection may overcome the basic states, locally and generally in a number of ways. An excessive virulent infection such as Vincent's infection may on occasion, dominate the local picture. The local and general basic states can on the other hand so deplete reserve that infection becomes established.

There are presented in clinical practice occlusions which are clearly the result of trauma and occlusions which produce trauma. As stated in an earlier paper (7), "in applying to the first class the term 'traumatic' and to the second class 'traumatogenic' in which the meanings conveyed are completely opposite, it would appear that terms have been chosen which designate these conditions more accurately thus making for greater clarity in dental nomenclature."

My definitions are as follows:

A traumatic occlusion is a contact relation of the masticatory surfaces of the teeth which is directly the result of trauma. A traumatogenic occlusion, as applied to natural dentures is an occlusion which under biting pressure produces an injury in the periodontal tissues.

Hanau (8) has defined articulation as the "change from one occlusion to another while the masticatory surfaces maintain contact." In other words, articulation is made up of a series of occlusions, occlusions being non-moving contact relationships of the masticatory surfaces. Where injury is produced in the periodontal tissues during the change from one occlusion to another while the masticatory surfaces maintain contact, a "traumatogenic articulation" may be said to exist. This appears to be in agreement with the viewpoint of Withycombe, (9).

My conception of traumatogenic occlusion, as recently stated, (10), involves a consideration of three main components, viz.—the mechanism, traumatogenic stress and the traumatic state produced in the periodontium. In other words, traumatogenic occlusion is a composite embracing these three elements, and appreciation of which individually and collectively is essential for an adequate understanding of the phenomenon. To the tooth and its supporting structures, I have given the term "dento-periodontal unit." The mechanism embraces two dento-periodontal units under biting pressure and involves the dento-periodontal relationship, including root-shape and socket-shape. It is well understood that a type of mechanism that frequently produces trauma is commonly found in unbalanced occlusion and articulation. That is to say, the occlusal contact of the dento-periodontal units of the mechanism is characterized by cusp interference with only too frequent introduction of excessive torque.

The term traumatogenic function by no means implies heavy function, and certainly is not synonomous with excessive function. It is apparent that greater care should be exercised in the use of the terms "overloading," "excessive function," "overstress" and "overfunction." So frequently one of these terms is used

when it is clear that heavy function within the limits of the reserve force of the cell is indicated. Confusion of meaning is still worse confounded when we see traumatogenic occlusion represented in the guise of a beneficial agent.

What then is the present status of traumatogenic occlusion as related to periodontal disease? It is hardly necessary to point out that according to my viewpoint as expressed in my writings, I believe it to be an extremely important factor in the production of Complex Periodontitis, primarily as a factor in the induction of early loosening, and secondarily, in its relation to gingival resistance to infection.

This viewpoint is amply supported by the observations of American and Australian writers. One need only call attention to the names of Stillman, McCall, Hutchinson, Smith, Tishler and Bricker of the United States, and Withycombe and Lewis of Australia, all outstanding clinicians. That the gingival tissues about teeth in traumatogenic occlusion are susceptible to infection is especially emphasized by Hutchinson, (11), Stillman, McCall, (12), and Withycombe, (13). Hutchinson has stated: "Constant sanitary treatment may preserve health, but practical immunity to bacterial invasion is achieved only by the removal of "all" causes of injury. Chief among these causes is malocclusion."

Further clinical evidence of a pericemento-gingival relationship is seen in the observations of Dunlop who has noted that the taping of the necks of teeth is followed by increased rigidity of the teeth in their sockets and marked increase in gingival tone and resistance to infection. Apparently the taping so vibrates the tooth in its alveolus that strengthening of the pericemental attachment takes place with a direct beneficial change in the gingival tissues.

Experimental proof is shown in the work of Lefcourt and Feldman, (14). A gold crown was inserted on the lower right first molar of a dog in such a way that there was occlusion only on that tooth and a "definite and typical Vincent's infection of the gingiva occurred which was most pronounced about the crowned tooth."

Traumatogenic occlusion is placed in a very subordinate role by the Vienna group. Gottlieb's conviction, (15), is that it cannot "cause loosening of the teeth" and from their experiments on dogs according to Mueller's summary, (16), he and Orban "emphatically concluded that in not one instance was it possible to show that pyorrhea or even an ordinary gingival inflammation had any causal connection whatsoever with traumatic occlusion."

It is apparent that the viewpoint held by the Australian, American and Canadian observers previously mentioned is incompatible with the Vienna concept.

Gottlieb holds that the basic lesion of genuine pyorrhea is a diffuse alveolar atrophy. This atrophy can localize about certain individual teeth in the denture, and "general affections, especially metabolic disturbances, must be held responsible." In other words, tooth-loosening as an early symptom in periodontitis is caused only by general systemic factors.

In pathology, the term "diffuse" is usually employed to mean "of undefined limits," and I regard it as a misnomer as applied only to one, or a limited number of sockets. An attack on the alveolar bone of only one tooth, even involving the entire socket, could hardly be construed as "diffuse," and it is not conceivable to me that general metabolic disturbances acting alone could produce it. It is hardly possible that a socket adjacent to an atrophied one, could itself be free from atrophy postulating that general agencies, only, are at work. A single atrophied socket, in the absence of local factors might be explained on the basis of an anomalous blood and nerve supply, which is of course not a general factor.

Cases are to be seen in every practice where looseness is manifested in a short time on abutment teeth, which were clinically tight to begin with, and in teeth in traumatogenic occlusion in finished orthodontic cases. These teeth soon tighten following occlusal adjustment. Is it conceivable that in the loosening and tightening, there occurred an attack and cessation of diffuse alveolar atrophy which coincided with the introduction and correction of traumatogenic occlusion?

My observations on the lower anterior teeth of sheep are possibly of interest. As Tomes has pointed out the "lower incisors are antagonized not by teeth, but by a dense gum which clothes the forepart of the upper jaw." In feeding, the blades of grass are grasped between the lower teeth and the pad and torn off by an abrupt movement of the head. In some sheep, the pad strikes the lower incisors only on the lingual surface of the crowns, producing through wear teeth that are chisel-shaped and extremely sharp. In others, the pad has a decided overjet, and in these cases, the pressure is mostly axial or end-to-end and the edges of the teeth are flat. In general, in different sheep of approximately the same age, the incisors of types under axial loading were tighter than those under oblique loading. And where the two classes of loading were manifested in individual sheep, the incisors obliquely loaded, that is to say under leverage, were markedly looser than the ones axially loaded.

Two different experiments were carried out. In one case, a gold crown was inserted, increasing the crown height, and thickness lingually in each case approximately one millimeter and a half, so that the brunt of the pad was first taken by the crowned tooth. In the other, certain teeth were ground down so that others first received the thrust of the pad.

Three months later, the clinical findings were as follows: With the exception of one tooth, which may be explained by peculiar tooth-pad biting conditions, all the overburdened teeth were decidedly looser. The measurements were made with the greatest possible care, under a magnifying glass, and with a mobility-tester especially devised for the sheep experiments by E. C. Platt. The mobility of the crowned tooth was increased three times with no increase of mobility of the associated lower teeth. And when pressed labially, a gross lingual pocket was evidenced, where with the same labial pressure, no pockets were to be noted clinically on the associated teeth.

In the other experiments, the overburdened teeth were markedly looser than their neighbors, but on labial pressure, no pockets were visible. Determination of pockets by probe-measurements was rigidly avoided owing to possible infectious complications from trauma. The microscopic findings will be reported at a later date.

My theory as to the origin of traumatogenic occlusion, in the main is based on the following points.

1. Hopewell-Smith, (17), in describing the skeleton of a man found in Essex in 1910 and computed to be 4000 years old, pointed out that "his teeth were worn flat and met edge to edge as in prehistoric races."
2. Keith's findings, (18), based on a survey of palates and jaws of British people, fifty ancient, (buried in England before the date of the Norman Conquest), and fifty modern, (chiefly Londoners of the eighteenth and nineteenth centuries), in which it is of interest to note that forty-seven of the dentures of the ancient skulls presented an end-to-end occlusion in the incisor region while all the modern skulls showed definite overlapping in the same region.
3. According to a recent report, (19), the inhabitants of Tristan da Cunha who largely have end-to-end bites, do not artificially clean their teeth, yet in the presence of tartar, periodontitis is rare.
4. Withycombe's findings, (20), on two large groups of children aged 12 to 14 years, in which he found only 1% having normal masticating occlusion, 99% being in traumatogenic occlusion.
5. Badcock's observations, (21), showed that fifteen years ago "German soldiers had far better formed jaws than English soldiers."

In the change from an end-to-end occlusion to one of overlapping there is implied a change from axial to horizontal loading in the anterior region, and with failure of the compensating curves to permit a balanced occlusion and articulation in the protrusive ranges, as is so often found, horizontal overloading is introduced, with, in so many cases, the factor of traumatogenic occlusion.

Traumatogenic occlusion, in this form, is, in general, of comparatively recent introduction, although as Keith has stated "Abiotrophic changes had overtaken the dental system of Western Europeans as early as the Neolithic period." "Form is the result of functional activity" (Lamarck), and as Badcock has pointed out "Nature, adapting means to need is reducing the jaws of civilized man accordingly."

Nature adapts or tends to adapt herself to changed conditions. "The whole process of inflammation is an exemplication of adaptation," Adami, (22). Many times the adaptation is far from perfect, and, in my opinion, the phenomenon of traumatogenic occlusion belongs in this category. It is only an incident in a background of profound changes affecting possibly the whole organism and certainly the teeth, jaws, nose, orbits and cheeks.

Brash, (23), has aptly said: "All this evidence not merely narrows the main field to a question which may be briefly characterized as one of Nature or early Nurture, but in fact strongly points to Nature rather than Nurture." . . . "An hypothesis that irregularity and malocclusion of the teeth are in a broad sense inherited conditions, that they depend upon genetic factors would seem at this stage to be a reasonable starting point and guide for further inquiry."

Prophylaxis, as Pitts, (24), has said, is merely applied etiology. The ideal denture is obviously represented in the end-to-end occlusion type, a result in common with short and broad dental arches and shallow palates, and I am strongly suspicious that, generally speaking, the various factors which produced these conditions, involved favourable periodontal adaptations to infection and traumatogenic occlusion as well. The disturbance of these factors, a concomitant of civilization, is undoubtedly largely dietetic and nutritional, and includes as well as its chemical composition, the texture, density and bulk of food.

Preventive measures may be divided into two classes, local and general. According to our knowledge of the local basic states, the normal ideal function is heavy function with increased stress within the limits of the reserve force of the cell. This is best accomplished through the establishment of balanced occlusion and articulation and it should be the objective of the dentist to strive for this goal where it is possible. In so far as this ideal is not achieved, either through lack of knowledge or skill, or because of the physical impossibility of the case, the tissues will with adequate circulation, adapt or tend to adapt themselves in like measure. The operation through which this is brought about is difficult, and demands a thorough working knowledge of the laws of occlusion, and articulation, and the mechanics of the "dento-periodontal" relationships. My views based on fifteen years experience are somewhat in sympathy with those of Withycombe and Taylor, (25), who believe that the operation is beyond the scope of the average dental practitioner.

Brushing, massaging and scaling are known artificial preventive measures, and the Dunlop Method, based on oxygen therapy, offers much promise.

General preventive measures have still to be tried on a fairly large group of individuals to afford any real test of their ultimate value. Agnew and Agnew, (26), in their nutritional experiments, found that in rats fed on a low calcium diet, periodontal lesions were found, whereas the controls were free. The lesions found varied in severity up to complete exfoliation of the teeth from the jaw. Marshall, (27), has shown in his studies of bone and tooth changes incidental to experimental tooth movement that "animals fed on deficient diets show a greater degree of resorption using the same appliances, than those on adequate diet." On account of great fundamental differences, the results of nutritional experiments on animals can not be applied too sweepingly to man.

Prevention of periodontitis for the community, as distinct from the individual under expert care, is as yet non-existent and the outlook is frankly

pessimistic but there is always the hope that science will unravel the mystery wherein by some short method ideal occlusions will be produced, with high local and general resistance to infection.

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Chronic Desquamative Gingivitis[1]

ARTHUR H. MERRITT, D. D. S., F. A. C. D., F. A. A. P.
New York City

Introduction and History

FOR a number of years I have been observing at infrequent intervals a relatively rare form of chronic gingival inflammation which because of its unusual and varying symptomatology is generally unrecognized or improperly diagnosed. To add to the confusion, it has been described with varying symptoms under several different names such as *chronic inflammation of the gums; chronic gingivitis and stomatitis; chronic diffuse desquamative gingivitis,* etc., with the result that its identity as a distinct disease has been obscured.

As early as 1894 Tomes (1) refers to a disease which he had observed as "a singular modification of chronic inflammation of the gums, in which, instead of becoming thickened and irregular on the surface, they seem rather to decrease in size and assume a very red, smooth and polished surface and mottled aspect." Goadby (2) calls attention to a disease which he describes as "characterized by bright red or scarlet gums and which commences insidiously by bleeding.—The disease shows no tendency to spontaneous healing and may remain present for many years.—The patient complains of great soreness, total inability to take any solid food when the disease is at its height, and even when the inflammation subsides any highly spiced or acid foods may precipitate another attack of ulceration." He says further that "in a well established case the whole of the mucous membrane of the gums and contiguous portion of the cheek, is bright red and injected.—The disease does not tend to be confined to the gum margins, but affects the whole of the gum tissue equally." Prinz (3) who has given the best description of the disease says that it "manifests itself by an excessive fungoid growth of the papillae, together with an intense inflammation of the marginal gum tissue within the vicinity of the necks of the teeth. The disease is primarily observed at the labio-buccal surfaces of the gingivae in both jaws: the lingual (palatal) region is rarely involved. It does not manifest itself in regions from which the teeth have been removed.—The highly edematous papillae present a deep bluish-red glazed appearance; their tips are frequently excoriated and they bleed on the slightest touch.—A mild offensive breath is always present. The patient complains primarily about great soreness and a burning sensation within the mouth especially during partaking of solid or hot food.—It is never observed in an edentulous mouth. The disease is primarily restricted to middle-aged women." In many respects this confirms my own observations. I have, however, never seen a case with any noticeable odor to the breath nor is it my observation that the lingual surfaces of the gums are only "rarely involved"; such involvement is less frequent but by no means uncommon. Moreover, the fungoid growths referred to by

[1]Read before the American Academy of Periodontology, Buffalo, N. Y., September 10, 1932.

Prinz have not been a prominent symptom in the cases I have seen. It is a disease that is probably more common than is generally realized for in its milder and less typical forms it may be confused with chronic gingivitis, as I diagnosed it in my earlier experience. During the past year I have seen no less than five typical cases in as many months, due in part to more careful diagnosis and in part to a larger number of referred cases.

Symptomatology

The most common objective symptoms as observed by me are:

First: An irregular inflammatory zone involving the gingivae and extending for several millimeters on to the gums. As a rule it does not involve all the gingivae, those most commonly affected being those of the maxilla in the incisor region. It varies in color from a bright scarlet to a bluish red, often having a mottled appearance. The demarcation between the healthy and diseased tissue is clearly outlined. The involved area frequently presents a polished glistening look. The bucco-labial surfaces are most commonly affected though it not infrequently involves the lingual surfaces also. I have, however, never seen it on the lingual surface only. It apparently appears first on the bucco-labial surface and by extension may then involve the lingual.

Second: Apparently the most characteristic symptom is that of desquamation by which the epithelial covering is sloughed off leaving a raw and sensitive surface; occasionally this can be removed in strips of considerable length. In some instances, at least, this condition is preceded by the formation of numerous small and slightly elevated blisters filled with a watery fluid. As they break and discharge their contents, they slough off leaving a raw and painful surface which in time heals over, only to repeat the same process later.

Third: Occasionally, though not as a rule in the cases I have seen, the septal gingivae take on a pedunculated fungoid appearance markedly different from that seen in hypertrophic gingivitis with which it cannot be easily confused.

Fourth: As I have seen it, it is always limited to the gingivae and adjoining gum tissues. It is never seen in edentulous mouths nor in those mouths in which it is present does it involve the gums where teeth have been extracted.

Fifth: I have never seen it except in women who have passed the menopause. Prinz reports having seen two cases in about a dozen in men past forty (3).

Sixth: It is extremely chronic and shows a disposition to frequent exacerbations. It may improve without any treatment only to relapse later, but it never entirely disappears. In those cases in which the gingivae become involved in fungoid growths these never clear up though they may at times be less troublesome. In typical cases there is always a history of periods of comparative comfort followed by relapses. Occasionally the cervical lymph nodes are painful to pressure. I have never noticed any odor to the breath. It may be found in mouths already affected by periodontoclasia as it may in those otherwise healthy. There is always

a complaint of soreness, and in periods of exacerbation, great discomfort in the use of hot or solid foods.

Etiology

Its etiology is unknown. I have exhausted all the resources of internal medicine and dermatology in an effort to get at the cause. Neither has thrown any light on the subject. Bacteriologic examinations have shown in some cases large numbers of fuso-spirochetal organisms, but since these are found in healthy mouths, they probably have no etiologic significance. The use of oxydizing drugs have only the most transitory effect on the disease. Goadby says it is usually associated with anemia and Prinz hints at endocrine imbalance, at the same time saying that nothing is definitely known about its causation.

Treatment

In mild cases limited chiefly to slight inflammatory reactions in which desquamation and hypertrophic gingivae are subordinate symptoms or absent altogether, the patient may be kept reasonably comfortable by the establishment and maintenance of good hygiene plus the application of such drugs as tincture of iodine, copper sulphate, 5% chromic acid, etc. Such treatment, however, will do no more than mitigate the symptoms; it will not cure and in some cases will give only the most temporary relief. In the more severe cases in which desquamation or fungoid growths are present, medication affords almost no relief.

Observing that the disease seems to be limited to the gingivae and adjacent gum tissue and that tooth extraction cleared up the trouble, I have been experimenting with the operation of gum resection which I am inclined to believe offers the only solution to the problem short of extraction. Prinz says that "the only successful remedy is the knife" and my experience, though at present limited, confirms this.

Having anesthetized the area to be operated upon, a horizontal incision is made on the bucco-labial surface well beyond the limit of the diseased tissue. If the lingual surface is also involved as is frequently the case, a similar incision is made on this surface, after which the gums and gingival tissues between the two incisions are completely removed exposing the alveolar process. In those cases in which no other disease is present, the new gum tissue will completely replace that which has been removed leaving almost no evidence of the operation and practically no recession. When, however, there is bone loss from periodontoclasia, there will be the inevitable recession which always attends gum resection in the treatment of these cases.

Immediately following the resection, the operated area is packed with a medicated cement such as that made by the use of oil of eugenol and commercial zinc oxide or the Ward pack may be used. This should be allowed to remain for a week or two by which time the new tissue will have largely replaced that which was removed. As soon as possible after removal of pack, the patient should begin and continue a regular system of massage of the gums for the pur-

pose of hardening and making them more resistant to disease. In very bad cases in which the teeth are seriously involved in periodontoclasia, extraction of the teeth may be the only remedy.

CASE REPORTS

Case No. 1.

Mrs. D., age about 55. First seen November 12, 1923 for treatment of periodontoclasia. Had already lost several teeth. Response to treatment fairly good. First noticed unusual appearance of gums on September 30, 1926. Smears showed large numbers of fuso-spirochetal organisms. Gums and gingivae dark bluish red. Marked desquamation which was preceded by the appearance of a group of small watery blisters. Bucco-labial and lingual surfaces were involved except where teeth had been extracted which were entirely healthy at all times. Complained of great discomfort. Referred her to a dermatologist who made a diagnosis of Vincent's infection. Anti-Vincent's treatment and prophylaxis afforded only temporary relief. Saw her more or less regularly for prophylaxis until the time of her death in 1930. Tried all kinds of medication without making any progress. This was one of the most typical cases I have ever seen, having all the classic symptoms, such as the periodic formation of watery blisters, marked desquamation, alternating periods of improvement and relapses, intractibility to treatment, and great discomfort at all times.

Case No. 2.

Mrs. H., age 53. First seen December 21, 1926. Most marked symptom was fungoid appearance of many of the septal gingiva. There was also redness of gums, discomfort in the use of the teeth with alternating periods of improvement and relapses. Both bucco-labial and lingual surfaces were equally involved except where teeth had been extracted. No history of desquamation. Medication and prophylaxis afforded but little relief. I saw her on an average of eleven times a year till 1931, during which time I experimented with every known drug that might possibly give relief with only temporary benefit. As her husband is a surgeon she had access to the best medical talent including two well known dermatologists, but without throwing any light on the subject. Finally on May 19, 1932 I persuaded her to make a trial of gum resection which was done on one side of the maxilla only with a view to subsequent treatment of the entire mouth if this proved satisfactory. By this time the patient was naturally skeptical of relief from any form of treatment, but in her extremity was willing to try anything that promised it. I saw her last on September 6; the side operated on was completely healthy and there had been no recurrence except slightly around the molar due probably to the fact that this tooth was more or less seriously involved in periodontoclasia and will probably have to be removed.

Case No. 3.

Mrs. P., age 58. Italian. Referred to me by Dr. Paul V. Clay of Binghamton, N. Y., on April 28, 1932. Examination revealed loss of four molar

teeth. There were no caries or periodontoclasia. Gums and gingivae were bright red involving both surfaces except about upper right molars and spaces from which the teeth had been removed which had never been affected. A few of the septal gingivae were edematous and of a bluish color; there was no fungus involvement. There was, however, a history of desquamation and great discomfort. The patient had been under treatment for several months without relief. A diagnosis of desquamative gingivitis was made and resection advised. This was done during May and June, 1932, removing completely the involved gums and gingivae on both bucco-labial and lingual surfaces leaving the alveolar process exposed. Cement was packed about the teeth so as to cover the entire operative area and allowed to remain for about two weeks after which it was removed and the patient returned to her home. In operating on one side a long strip of the desquamating epithelial surface came away as the horizontal incision was made on the lingual surface. When seen a month later satisfactory progress toward recovery was being made. In a letter dated July 29 she reports "that my gums have improved greatly and I am feeling much better." It is too early to make a final report in this case. To date she has made a very satisfactory progress with every promise of permanent results. In a later letter dated September 23, 1932 this patient writes, "I am glad to report that my gums are coming along very nicely. There are no spots on them nor do they trouble me. I am very grateful to you for succeeding in curing me."

* * *

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The Dynamic Relationship Between Pathologically Migrating Teeth and Inflammatory Tissue in Periodontal Pockets: A Clinical Study[1]

By Isador Hirschfeld, D. D. S.
New York City

OF THE irregularities of the dental arch associated with periodontal disease, some may be, as is well known, the cause of the disease, others, the effect. One instance of the latter is that brought about by the pathologic drifting of a tooth from its original position and location, as a result of the expansion pressure of growing granulomatous tissue in a pyorrheal pocket, on that side of the tooth from which it is being pushed. This has been noted in dental literature on several occasions but in no instance, to my knowledge, was there any allusion to the probability of a favorable prognosis, and one writer ventured the statement that such teeth, even if treated, must sooner or later be extracted.

The principal aim in this paper is to point out, by means of illustrative cases from practice, not only that such teeth can frequently be made permanently healthy but that as a further result of treatment, they are very apt to return to their original position and location without any orthodontic aid. A definite knowledge of this favorable prognosis is of utmost value and of sufficient importance to merit particular emphasis.

The following case is a typical instance of the extreme migration of a tooth away from its normal position. Fortunately, I was able to secure an authentic photograph of the normal arch prior to its disturbance by the pathologic forces.

Case I. Mrs. L., age 34, housewife, gave a history of bleeding gums since approximately the beginning of adolescence. She presented at the clinic with a generalized, advanced pyorrheal condition, including many teeth of negative prognosis, especially the upper left central incisor which had a mobility degree No. 3 (on the basis of 1, 2, 3). Although a deep pyorrheal pocket partly encircled it on its labio-mesio-palatal sides, it was only on the mesio-palatal aspect that a large mass of granulomatous tissue, protruding somewhat out of the pocket, was prominently evident, and obviously the cause of the tooth's migration in the opposite direction, namely, disto-labially (Fig. 1). The patient volunteered the information that as a girl she had a perfectly arranged set of teeth. At my request, she brought a photograph taken at that time, which corroborated her statement (Fig. 2). However, it showed, in addition, hypertrophied septal gingiva between the upper central incisors. The periodontoclasia was not treated, as extraction was considered more expedient, for practical purposes, but the case is presented because

[1]Read before the American Academy of Periodontology, Buffalo, New York, September 10, 1932, as a contribution to the annual report of the Committee on Scientific Investigation.

of the unusual photographic record and also as additional evidence of my belief, as stated in a recent paper, that a chronic hypertrophic gingivitis occurring in youth, if permitted to continue untreated, tends to eventuate in the adult, into chronic Vincent's infection or into suppurative periodontoclasia[1].

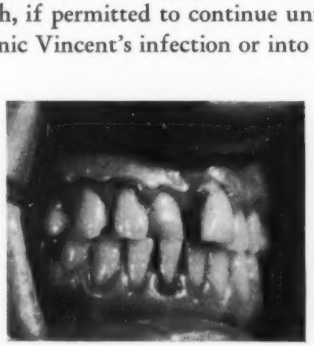

FIG. 1

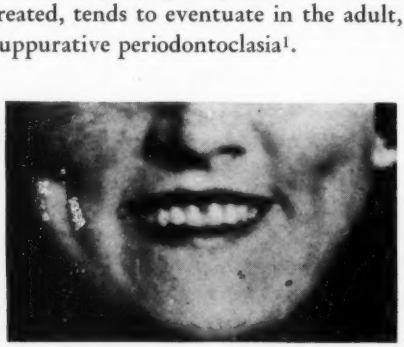

FIG. 2

FIG. 1. Case I (see text), showing the migration of the upper left central incisor disto-labially, caused by the expansion pressure of granulomatous tissue in the pyorrheal pocket on the mesio-palatal side of the tooth. Note the drop of pus expressed at the time of photographing.

FIG. 2. Same as FIG. 1. Enlargement (made with the aid of a microscope) of a very small picture of the patient taken 18 years earlier. The teeth were in approximately normal alignment, but the pathologic (hypertrophied and red) condition of the central papilla was quite evident.

The next case is one of many instances in my practice which demonstrate that it is possible to obtain, through treatment, the return of the migrated tooth to its original position, unaided by orthodontic means; also that it may remain so permanently.

Case II. Miss N., age 23, school teacher, was advised by a periodontist to have all the upper incisors extracted, not only because they had deep pyorrheal pockets but especially because the lateral incisors were decidedly out of alignment (Fig. 3). He considered them unattractive and of comparatively poor prognosis; therefore, not worth saving. The patient, unwilling to take this advice as final, came to my office for another opinion. It was evident that the etiology of the abnormal position of the teeth was similar to that above described; accordingly, I informed her that the teeth could not only be made well but that they would probably return to their original position with the attainment of periodontal health.

The main complaint, in addition to the generalized pyorrheal infection, was the recent, very rapid drifting of the upper right lateral incisor out of the arch. The subgingival inflammatory tissue in the deep pyorrheal pocket on the mesio-palatal side of that tooth pushed it in the diametrically opposite direction, namely, disto-labially, in pendulum like fashion, the apical end of the root acting as a fulcrum. Although the tooth had been dislocated to a marked extent, the degree of the mobility was but slight in proportion. It could be moved from its *acquired* centric position in the centrifugal direction to the extent of degree No. 1, and in a

centripetal direction, that is, towards its original position, ½ degree, giving a total mobility of degree No. 1½. (It is interesting to observe that this approximate ratio of 2 to 1 seems to obtain in most teeth so dislocated.) The tooth stood far out of occlusal contact with its opponents in both the centric and eccentric positions of the mandible; therefore, although traumatic occlusion may have been an etiologic factor in the establishment of the original periodontal lesion and possibly in the initiation of the migration, it could not have been directly responsible for the further drifting of the tooth. The left lateral incisor which also had a pocket on the mesio-palatal side of the root was similarly forced in a disto-labial direction but to a lesser extent. The central incisors had pockets on their distal sides and were accordingly wedged tightly against each other.

The usual favorable results were achieved by the routine conservative treatment for suppurative periodontoclasia. However, it is interesting to follow the progress of the right lateral incisor. As it stood completely out of occlusal contact with, and far from, its opponents, and as it presented no morphologic abnormalities, no grinding was at first indicated. But, with the resolution of the granulomatous tissue, it soon moved into occlusion and then actually into traumatic occlusion because it had become somewhat elongated and the cingulum interfered with its complete return to the original position (Fig. 4); therefore, corrective grinding was now necessary. The degree of mobility remained unchanged during the return migration and the tooth became normally firm some time after it resumed its normal position (Fig. 5).

FIG. 3

FIG. 4

FIG. 3. Case II—showing the upper right lateral incisor pushed away disto-labially from its normal position and out of occlusal contact, by inflammatory tissue in a pocket on the mesio-palatal side.

FIG. 4. Same as FIG. 3—one year later, after successful treatment of the pyorrheal condition, the tooth drifted back to its original position, into contact with the central incisor.

The return movement of the other teeth was uneventful. At the present time, 6 years later, the teeth continue to maintain their position and periodontal health. The labial gingiva which had receded somewhat before treatment, grew down with the tooth in its slight elongation, thus retaining evidently its relative

position since, according to the casts, there was no indication of detachment or further recession, when the return migration was completed.

FIG. 5. Radiographs of Case II—Left, before treatment, right, one year later.

Comments: 1. That the motive power in the outward migration of the tooth was furnished by the expansion force of the subgingival inflammatory tissue in the pocket on the opposite side of the tooth seems borne out by the facts; *a*, the return movement of the tooth was in proportion to the rate of shrinkage of the inflammatory tissue soon after the beginning of treatment; *b*, the tooth moved very rapidly at first but later more slowly which was the exact reverse of the outward migration which was slow at first, then very rapid; *c*, the rapid return of the tooth was accomplished without any special effort by the operator to minimize any possible occlusal forces, thus indirectly demonstrating that masticatory or other occlusal forces could not have been directly responsible for the migration. 2. The degree of mobility is not necessarily in proportion to the extent of the migration; it may be very slight even though the tooth has drifted considerably.

The case that follows is an outstanding example of the possibility of the wholesale migration of teeth in one mouth and their automatic return as a result of routine periodontal treatment.

Case III. Miss B. S., age 19, presented with an unusually advanced condition of suppurative periodontoclasia, considering her youth. Most of the teeth were affected (Fig. 6). They showed deep pyorrheal pockets and were widely separated from one another (Fig. 7), the interdental spaces having been produced in no small degree by the growing masses of subgingival inflammatory tissue in the pockets and by the hypertrophy of the interdental gingivae. As a result, many of the teeth assumed an inclined position in the labio-buccal direction, thereby broadening the arches materially and disarranging their occlusion.

Ordinarily, such marked destruction of alveolar process as indicated by the X-rays, would prompt a questionable or negative prognosis and extraction of the teeth would probably be advised. Treatment was undertaken only at the insistence of the patient and her parents. The teeth soon began to move closer together, so that the arches automatically approached their original size (Fig. 8).

In this case, too, the return movement, though rapid at the beginning, soon slowed down to such an extent that the migration still continued after the periodontium had become well. Comparative measurements of the pre-operative and post-operative casts (Fig. 9) showed a shortening of from two- to three-sixteenths of

Fig. 6. Radiographs of Case III before treatment.

an inch, of the various diameters of the arch, and but few interdental spaces. (Knowledge of such and other anatomic changes as a result of periodontal treatment should guide us in the timing of the insertion of restorations according to the individual requirements.) The mouth continues in a healthy and useful condition through the excellent home care by the patient.

FIG. 7

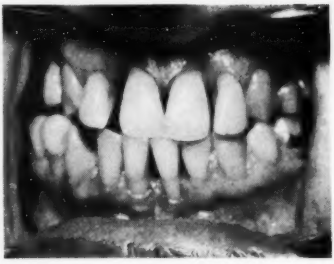

FIG. 8

FIG. 7. Case III—Photograph made before the beginning of treatment. Note the marked labial incline of the anterior teeth and the numerous spaces between them.

FIG. 8. Case III—Several years after treatment. Note the complete elimination of some of the interdental spaces and the marked reduction of others. The teeth have more nearly approached their normal incline and position in the arch.

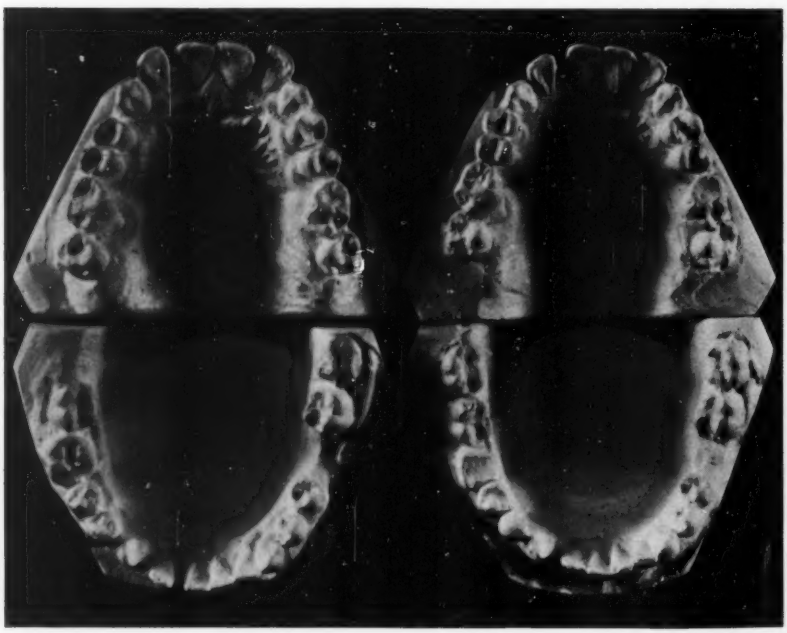

FIG. 9. Occlusal view of casts of Case III—Left, before treatment; right, several years later. Both arches became between two- and three-sixteenths of an inch smaller in every diameter.

The following two case histories serve to illustrate that subgingival granulomatous tissue may, in some instances, act merely as a prop on the diseased side of a tooth largely deprived of supporting alveolar structure. Consequently, the diminution in size and final disappearance of this tissue with the advent of periodontal health, is followed by a corresponding tilting of the crown end of the tooth in the direction of the space formerly occupied by it, unless the alveolar process regenerates sufficiently in the meantime to prevent this. Such a possibility in the prognosis must be guarded against and forestalled by means of permanent or temporary splinting so that the tooth may be maintained in its original position until the alveolar process has been given an opportunity to regenerate to its maximum. Otherwise, two adverse results are likely to accrue: an unesthetic appearance and a tendency to the re-establishment of periodontal disease through traumatic occlusion or leverage.

Case IV. Miss C., age 24, presented with a generalized advanced suppurative periodontoclasia. The upper central incisors, especially, were badly affected, the right one being of mobility degree no. 2½ and the left, no. 2. Each of these had deep pockets on the mesial side, the one on the right central being very marked mesio-palatally. A large space separated that tooth from the lateral incisor and another somewhat smaller, from the neighboring central (Fig. 10). The patient remembered clearly that the latter space had always been there: therefore, it was not a result of the presence of inflammatory tissue, which in this case evidently acted only as a prop in lieu of the destroyed alveolar process. As a result of treatment, the right central incisor migrated in the mesio-palatal direction until it overlapped the neighboring tooth on its palatal aspect (Fig. 11); in other words, it had moved directly to the side where the pocket had had the greatest depth and

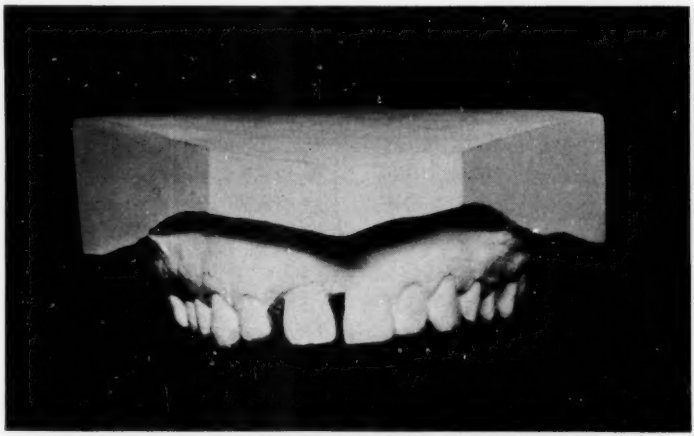

Fig. 10. Case IV—Cast made before the beginning of treatment, showing a large interdental space between the upper central incisors.

into the space left by the resolved granulomatous tissue (Fig. 12). In its new position, the tooth was being greatly traumatized by the opposing teeth. The corrective grinding at this point evidently permitted the lip to force the tooth still further in the lingual direction. The wisdom of fixing such a tooth by means of mechanical intervention is evident.

FIG. 11

FIG. 12

FIG. 11. Same as Fig. 10, photographed two years after treatment.
FIG. 12. X-rays of Case IV—Left, before treatment; right, after treatment.

Case V. Mr. L., age 37, had a generalized pyorrheal condition, especially marked in the lower anterior part of the mouth, the right central incisor being of mobility degree no. 2, the left one, no. 2 plus, and the left lateral, no. 2. The mandibular median line had moved towards the right side through the drifting of the teeth on account of the early loss of the first molar on that side (Fig. 13). Although the left central incisor had crossed the median line, thereby causing a large open space between it and the left lateral incisor, it had not quite come into contact with the right central. The X-ray (Fig. 14) shows greatest alveolar destruction on its mesial side, and the record chart, too, indicates that the pocket there was very deep especially on the labial aspect. With the elimination, soon after the beginning of treatment, of the granulomatous tissue which had acted as a prop, the tooth, now deprived of its support, was permitted to tilt over into the space left by it, and into contact with the remaining alveolar process which had only partly regenerated, although it was now healthy. When the patient returned after an absence of about a year, it was found that the tooth had inclined still further in the direction where the pocket had been deepest, markedly overlapping labially the neighboring central incisor. This extreme tilting might have been prevented through prosthetic interference had the patient come earlier.

The dynamic force exerted by the expansion pressure of the subgingival inflammatory tissue may, in some instances, be sufficiently great to cause the migration not only of the tooth affected by the periodontoclasia, but also of its possibly larger neighbor. Such an occurrence may, in addition, give rise to the subjective

symptom of a feeling of interdental pressure not unlike that produced by food impaction although no food is actually present, as demonstrated in the following case:—

Case VI. Dr. R., age 30, physician, presented a more or less generalized pyorrheal condition. The mouth showed a rachitic picture of hypoplastic enamel and anterior non-occlusion, particularly of the incisors which were considerably denuded of periodontium. According to the diagrammatic examination chart, all showed some pyorrheal pockets except the upper right lateral. The mobility of

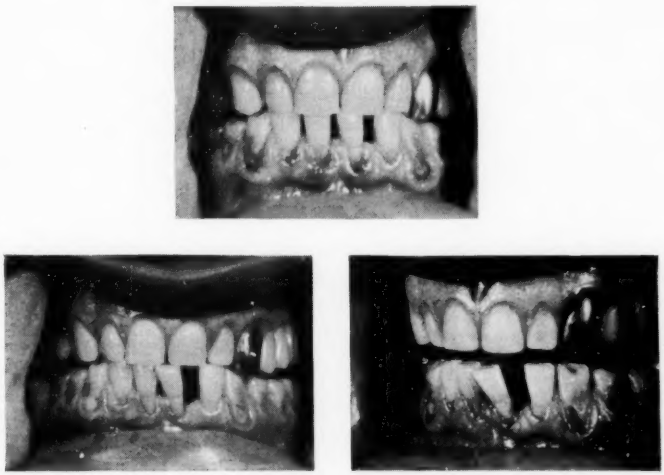

FIG. 13. Case V—Above, before the beginning of treatment.

Below—Left, three months later, after treatment. Note the marked improvement in the gingivae, also that the lower left central incisor has at this time come into contact with the right central, slightly overlapping it. (See FIG. 14.)

Right—One year later. The lower left central incisor is tilted over considerably in front of its neighbor. The patient had neglected to return for follow-up inspection and prophylaxis. Although his mouth was kept in an unhygienic condition and there was a slight gingivitis, suppuration had not returned, evidently indicating that the treatment for pyorrheal infection was successful.

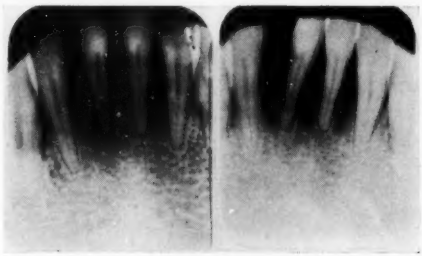

FIG. 14. X-rays of Case V. Left, before treatment; right, after treatment.

these teeth was no. 1½ with the exception of the upper lateral incisors which were slightly firmer. After apparently satisfactory response to treatment, the patient was discharged and requested to return semi-annually for inspection and prophylactic treatment.

Four years later, after an absence of a whole year, he called with a complaint of an annoying, continuous pressure between the upper right central and lateral incisors as if caused by food impaction in spite of the fact that he was unable to find any food. A comparison of the mouth with photographs and casts made at the beginning of treatment showed that whereas these two teeth were very far apart at that time, they had now approximated into actual contact (Figs. 15 and 16). Further examination disclosed a deep, newly formed pyorrheal pocket on the disto-labial aspect of the lateral incisor, and true to form, that tooth was forced in the mesio-palatal direction; but more than that, it pushed the neighboring central incisor, in billiard like fashion, mesio-labially, to an appreciable degree. The patient was about to leave for his summer vacation and was given but one treatment. However, he later informed me that the single treatment (which consisted of subgingival curettage) had relieved the pressure considerably. Evidently, it was reduced through the resultant resolution of the inflammatory tissue. Unfortunately, I have been unable to follow up this case further and cannot report the ultimate outcome.

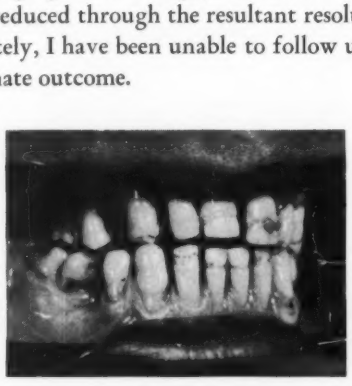

FIG. 15

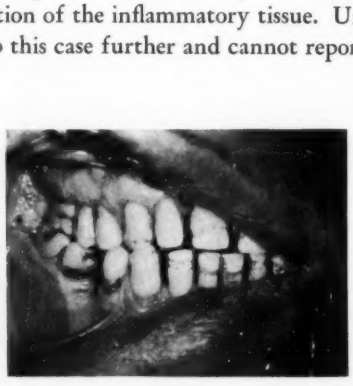

FIG. 16

FIG. 15. Case VI, showing the upper right lateral incisor in close contact with the central incisor. The patient had a feeling of pressure between these teeth as if produced by food impaction, but there was no evidence of food. See FIG. 16.

FIG. 16. Same as FIG. 15, photographed prior to the treatment for generalized suppurative periodontoclasia four years earlier when the upper right lateral incisor was evidently not involved. Note the large space between the upper lateral and central incisors. (This photograph had been taken simply to record the frenum between the lower cuspid and first bicuspid with relation to a special study the author was then making, but incidentally, it gives a record of the position of the teeth at that time.)

Comment: In an attempt to investigate the etiology of the new pocket formation, I found, upon inquiry, that the patient had recently acquired the habit, during office hours, of holding his fountain pen between the embrasures formed by the incisal edges of the upper and lower incisors and cuspids on the right side. It is possible that this was done as a reaction to an uncomfortable feeling in that

area, and that the periodontal disease was thus indirectly aggravated by the incidental traumatic occlusion. Guided by previous observations on the dynamic relationship between a pathologically drifting tooth and subgingival inflammatory tissue, I looked for and found the deep pyorrheal pocket on the diametrically opposite side of the tooth. The diagnosis of the etiology of the subjective symptom was evidently corroborated by the immediate relief obtained from the treatment.

Up to this point the cases described dealt with the pathologic drifting of the crowns of the teeth. However, a reverse process is evidently possible, in which the root ends migrate from their normal position while the crown ends remain comparatively stationary, as indicated by the following case history of a young adolescent.

Case VII. Master S. R., age 14½, notwithstanding his extreme youth, presented with a symmetrically distributed, very advanced pyorrheal condition. At the age of 12½, 2 years before I saw him, he was already forced to seek aid for periodontal ills, but with no tangible results, as the edematous condition of the gum and the copious flow of pus had not been noticeably lessened (Fig. 17). His medical history showed that he had been malnourished and sickly since, and probably before, birth. Many of the systemic symptoms of which he complained were evidently those of nutrition deficiency. This diagnosis was further established by the record of the patient's food which indicated that he ate no vegetables except green peas, and little of fruits, which consisted mainly of apples, bananas or plums and very rarely, an orange. Treatment of his condition began to show results only after he was placed on an improved diet (Fig. 18).

Fig. 17

Fig. 18

Fig. 17. Case VII, showing evidence of suppuration and deep pyorrheal pockets between the upper central and lateral incisors and between the lower lateral incisors and cuspids. The pockets were filled with masses of granulomatous tissue which caused the alveolar gingiva to bulge out.

Fig. 18. Same as Fig. 17, after treatment. Note the thinning down of the alveolar gingiva which had formerly bulged out. This, no doubt, was due to the disappearance of the large masses of granulomatous tissue in the subgingival pockets as a result of successful periodontal treatment.

The patient presented deep pyorrheal pockets between the lower lateral incisor and cuspid on the right and left sides of the jaw with the gingival tissue between these teeth distended by a growth of subgingival granulomatous material. X-rays of these parts showed that the roots of both lateral incisors inclined towards

the centrals with their apices almost touching, thus producing a fan shaped group (Fig. 19). Intermediate serial X-rays taken after treatment of the right side, but before serious local work was undertaken on the left, showed that the right lower lateral incisor had returned to its original position, but the left only partly; also, the regeneration of alveolar process at these points showed an asymmetrical rate of progress in both the upper and lower jaws on the respective sides. A parallel asymmetric clinical improvement was then also apparent. It was evident from progressive X-rays that as periodontal health returned, the left incisor, too, was moving towards its normal position, the four roots thus finally becoming parallel.

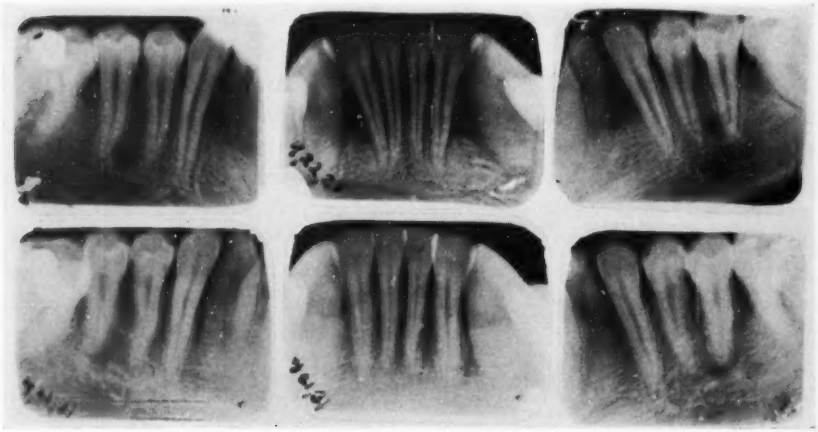

FIG. 19. X-rays of Case VII. Above, before treatment. Note the fan-shaped alignment of the lower incisors. Below, after treatment, three years later. Note the extent to which the apices of the lateral incisors moved away from those of the central incisors. The cuspid-bicuspid regions are also shown for comparison, to eliminate the possibility of an optical illusion caused by faulty technique in radiographing the incisors; these X-rays were taken at approximately the same angle, but whereas in the pre-treatment films, the teeth stood far apart, they are closely approximated in the post-operative films, thus corroborating my assumption that migration had actually taken place.

Comment: The root ends of the lower lateral incisors had probably been forced towards the centrals by the expansion pressure of interproximal inflammatory tissue between the lateral incisors and the cuspids. The crowns were very tightly interlocked and could not readily be moved from their position; therefore, the interdental pressure forced the root ends instead, the bone in their path succumbing, probably, to pressure atrophy. This explanation seems tenable since the bone at this age is in a formative stage and very little pressure is required, as in orthodontic treatment, to make it yield.

As the original photograph indicates, the expanding inflammatory tissue, in addition to pushing the roots apart, also pushed the gingiva into corpulent contour.

In trying to account for the return of the teeth to their probably original position, I can only suggest the possibility of a combination of a form of leverage

produced by occlusal pressure in mastication and the pressure produced by growing forces in young bone.

Summary

1. Clinical experience, supplemented by accurate records, indicates definitely, notwithstanding statements to the contrary, that teeth pushed away from their original position by the expansion pressure of subgingival granulomatous tissue may not only be made permanently healthy, but that they may, without any orthodontic aid, also return to, and remain in, their original position in the arch.

2. The degree of mobility of such teeth is not necessarily proportionate to the extent of the dislocation.

3. In the cases observed by me, there seems to have been a constant ratio of two to one between the degree of mobility from the acquired centric position in the centrifugal direction and that from the same point in the centripetal direction.

4. During the automatic return of the tooth, the degree of mobility seems to remain unchanged until some time after the tooth has reached its original position, when it may become normally firm.

5. Although traumatic occlusion may, in some instances, be a factor in the establishment of the initial periodontal lesion, it does not necessarily continue so during the migration of the tooth; accordingly, such a tooth may automatically return to its original position without any attempt by the operator to modify the occlusion.

6. In some instances, the subgingival inflammatory tissue may act merely as a prop to a tooth greatly deprived of supporting alveolar structure, and therefore, with the resolution of this tissue as a result of treatment, the tooth tends to tilt into the space left by it.

7. Through accurate record casts, it may be observed that not only individual teeth, but entire sets, may change their position in the arch as a result of successful periodontal treatment. It is obvious, therefore, that the insertion of post-operative restorations should be timed in accordance with individual requirements.

8. Under certain conditions, the expansion of the inflammatory tissue may produce a feeling of pressure between two teeth, not unlike that experienced from the impaction of food. Such a subjective symptom when unaccompanied by the actual presence of food should, therefore, suggest this etiology.

9. The writer suspects that even as the crown ends of teeth may be forced from their original position by subgingival granulomatous tissue, the apical ends, too, may be thus displaced while the crowns remain comparatively stationary.

REFERENCE

1 Hirschfeld, Isador, "The Toothbrush in the Treatment of Chronic Hypertrophic Gingivitis", Dental Items of Interest, pp. 412 and 428, June, 1932.

730 Fifth Avenue.

Some Clinical Phases Of Nutritional Problems In Periodontoclasia[1]

By AUSTIN F. JAMES, D. D. S., F. A. A. P., Chicago, Illinois

IN SO far as we now know there is no laboratory test that would determine a lack of vitamin C in the diet. Much has been written about the calcium and phosphorus content of the blood and saliva, but there is no proof and little indication that any correct conclusions have been drawn where gingival disturbances are encountered; nor have they been proven a factor in dental caries, though there is an attempt to make this latter association.

It is invariably true that patients suffering from gingival disturbances (and we believe it to be equally true of dental caries) are eating too largely of carbohydrates and proteins and insufficiently of raw green vegetables and fruits. Their meals usually are as follows: Breakfast—oatmeal (or other cereal), sugar, cream, toast or rolls, and coffee. Luncheon—a meat sandwich, or rolls and coffee. Dinner—a hearty meal of meat, bread and potatoes, eaten to the point of satisfying hunger; vegetables or fruits are consumed only as appetite dictates and often are omitted.

The non-vitalistic theory of dental caries is to the effect that when teeth are once formed and in position in the arch, they have no power of resistance. If they are kept clean, teeth will not decay and if they are not kept clean they will decay. According to the vitalists, however, teeth can, in some way and under some conditions, protect themselves against invasion.

During forty years of the practice of dentistry I have followed and practiced the non-vitalistic theory in regard to dental caries. I still believe that everything possible should be done to keep the mouth and teeth clean; and have not lost faith in the value of gum massage. But in those forty years of practice I have been converted to the vitalistic theory and now believe that caries and gingivitis are an indication of systemic and nutritional disturbances. This is the only way to explain why it is that many extremely unclean teeth do not decay and why it is that many clean teeth decay rapidly. A sick person does not need a physician or laboratory test to tell when he is well; his whole being tells him that.

We see beautiful sets of teeth and healthy gums and yet have no way of telling why this is so, but are expected to explain why the reverse is true. Bacterial theories of dental decay and gingival infections are not disproven, but they are invariably secondary in their importance.

About nineteen years ago a group of twelve dentists in Chicago formed a study club, but through ignorance of what the word *research* really meant, called themselves the Chicago Dental Research Club and obtained a state charter. The most remarkable thing about this group is that the members of it have remained

[1]Read before the American Academy of Periodontology, Buffalo, New York, September 10, 1932.

together all these years, attended every session, and kept up their obligations. The Club has been the means of keeping in touch with modern developments in pathology and other important subjects.

After taking up a study of the chemistry of life and following it in a general way through digestion, nutrition and assimilation, we became especially interested in nutrition and its relation to the pulps of teeth and their supporting structures. Animal experimentations by Professor Milton T. Hanke, our co-worker and instructor, as well as the work by many others throughout the world on this subject, have supplied convincing proof that diet and nutrition are important factors in general health.

What we are desirous of presenting is the question, "Can the clinical observations of twelve men working together on the same subject constitute a research?" We are thoroughly convinced that we are obtaining and maintaining lasting results by increasing vitamin C in all forms of gingival disturbances that are not obviously due to mechanical or un-hygienic complications. The only thing that is new in our findings is the amount of vitamin C which we believe is necessary to correct an obviously vitamin C deficiency.

Returning to the bacterial aspect of the question where there is infection, it is not different from that found anywhere else in the body. Cleaning and draining the area and reducing to a minimum the number of bacteria, we get rid of the infection. Obliterating the pockets by gum massage with the toothbrush and building up resistance by the squeezing process keeps down bacteria and prevents re-infection. But this has not been enough. We were never sure of not having recurring gingivitis to a greater or less degree.

Since we have been prescribing the larger amounts of citrous fruit juices and in some cases increasing the milk and egg intake, more lasting results have been obtained. We are never sure of this, however, under three months rigid application of this modified diet. These patients invariably report a glowing sense of energy and well being, and are enthusiastic adherents of the program. Every dentist in the group recognizes in these patients an orange tinge or glow in the complexion: they represent the picture of health.

The general agreement of these findings in a large number of patients may not constitute sufficient proof, but the careful and critical manner in which the observations have been made, certainly places them on a high plane of clinical research. When expressing regret to Dr. H. Gideon Wells, the head of the research department of the Sprague Memorial Institute at the University of Chicago, that we had insufficient laboratory proof, he said, "Clinical findings are always the final resort in the study of any health problem." We need no laboratory test to convince us of health, either in person or in observable tissues. This being true, it has seemed that the study and general agreement of twelve men over a period of years is at least worthy of recording and may perhaps find corroboration from other workers.

We have attempted in advanced cases of periodontal conditions to determine whether or not we really obtain a laying down of denser bone following our efforts to increase calcium metabolism. But again, the most convincing proof that we have been successful are the clinical findings. The teeth seem firmer in their sockets, but so far the X-ray findings have been far from convincing.

In conclusion I wish to present the list of foods which Professor Hanke believes should contain all of the substances that are required to build healthy body tissues. Also to add a few case histories which bear out his findings.

Eight ounces of orange juice and the juice of ½ lemon once or twice daily, depending upon the age and gingival condition of the patient. Patients with acute ulcerative gingivitis receive a quart of orange juice and the juice of four lemons a day for ten days or until the acute condition has subsided. Also eight to 32 ounces of milk. Children are given 32 ounces; adults usually not over 8 ounces and 1 or 2 eggs. Adolescent children and pregnant women receive 2 eggs; one quarter to one-half head of lettuce; one-fifth of a pound of fruit besides the orange juice; 0.8 to 1.0 pound of vegetables; meat once a day. Other foods as desired.

Case Reports

Miss J. G., seventy-two years of age, has been my patient for twenty years with always recurring gingivitis and a great amount of both salivary and serumal deposits of tartar. Prophylactic treatments were given four times a year to maintain gingival control. Patient had neuritis in left arm and shoulder and general pains throughout the body.

Two years ago I tried to persuade her to take the large amount of orange juice, but with no success until she told her physician. He advised her to follow my suggestion. For a year and a half she has continued to take the full pint of orange juice daily. June 20, 1932 the patient enthusiastically reports complete freedom from neuritis in arm and shoulder, as well as the disappearance of a general body soreness that had bothered her for years.

The gum condition is almost perfect—no salivary deposits, and almost no serumal; only a few small nodules are found in the interproximal spaces in the molar regions where there has been loss of alveolar bone, making it difficult at these points to keep them clean. She is so much freer from constipation than ever before that she reports a most unusual and satisfactory sense of well being. She believes that the orange juice is responsible for her improved condition.

Mrs. J. C. M., age 38, presented for treatment November 12, 1928, with a very advanced case of periodontoclasia but no caries. The pockets were deep and badly infected. Patient had been advised to have all teeth extracted. Height 5′ 3″, weight 100 lbs. Ca 10.85. P. 2.57 (serum). r.b.c. 4,240,000. w. b. c. 8,000. b. p. 94/60.

The teeth were thoroughly scaled, the technique of gum massage was taught and the occlusion balanced by grinding. The diet outlined above was recom-

mended and has been conscientiously followed up to the present time. Weight had increased to 120 lbs. on June 6, 1929. General health excellent. Gingivae healthy, no pus and only two teeth loose. The last laboratory check was made July 3, 1930 which gave her weight as 123 lbs., Ca. 11.21, P. 3.39 (serum), r. b. c. 4,300,000, h. b. 12.7, w. b. c. 7,600, b. p. 94/60. General health had remained excellent in spite of considerable mental distress. Gingivae healthy, no pus and only one tooth (u. l. 1st bicuspid) loose. Patient was last seen in July, 1932. General health has continued excellent. Gingivae healthy. No pus. Upper left first bicuspid remains loose but does not interfere with comfortable function.

Mrs. A. M. D., age 38, first presented November 2, 1926 with a beautiful set of teeth in good alignment but with very prominent cusps on molars and bicuspids showing no wear from function. Advanced pyorrhea with deep pockets and pus in all bicuspid and molar regions. Condition November 29, 1929 was still one of an active pyorrhea. Ca. 9.8. P. 3.15 (serum). r. b. c. 4,670,000. w. b. c. 5,950. Dietary regime had been instituted March 29, 1929, but had, up to November, led to little, if any, betterment in the oral condition although the patient's general health had apparently improved. Improvement in the oral condition has been slow and continuous up to the present time. The mouth has been free from pus for over a year and there is no evidence of a relapse between visits.

Mrs. W. P. H., age 59, presented June 8, 1916 with an advanced case of pyorrhea. There were numerous loose teeth, deep pockets, and considerable pus. The upper left cuspid was very loose and twisted to three-quarter turn labially. This tooth was returned to its normal position and held in place with a retainer, but would not remain in position without a retainer. Patient travels a great deal and was seen at irregular intervals. Always responded to prophylactic treatment and always returned with teeth loose and pockets infected. Dietary regime was instituted January 27, 1929. Height 5′ 5″. Weight 155 lbs. Ca. 10.7. P. 3.81 (serum). r. b. c. 4,240,000. w. b. c. 7,300. hb. 12.0 g. b. p. 165/105. Re-check on October 13, 1930, gave—weight 155 lbs., Ca. 11.48. P. 3.57. r. b. c. 4,340,000. hb. 13.4 g. w. b. c. 6,950. Gingivae healthy. No pus. Teeth tight. Patient has continued to enjoy oral health up to the present time. The upper left cuspid has remained in position without a retainer.

Mrs. C. M. P., age 50, has been afflicted with spongy bleeding gums for many years. Prophylaxis has been carried out three to six times a year without any appreciable amelioration of the condition. The gingivae have been congested and highly inflammed; but pus has never been present. Patient is of a highly nervous temperament and tends to be dejected. I attributed her oral condition at least partially to her nervous temperament and did not believe this to be a case that could be benefited by dietary means. She was, however, placed upon the dietary regime November 9, 1930, and has co-operated very well. The gingivae have become less thick, and certainly can not be called spongy; they seldom bleed. A complete cure has not been obtained; but the results have been very gratifying.

The Place Of Dentistry And Periodontia In The Social Scheme[1]

By SIDNEY J. RAUH, D. D. S.
Cincinnati, Ohio

CAN YOU answer this question? Shall dental service develop into a luxury to be enjoyed only by the wealthy?

It is fairly definitely understood that not more than twenty to twenty-five percent of the people of the United States receive regular dental attention, and the rest of the world far less.

It would be of value to consider who constitute this twenty to twenty-five percent; from what classes do they come? For many years the better educated have taken care of their teeth to some extent, and Booker T. Washington's statement "civilization follows the toothbrush" was uttered only a quarter of a century ago. There are, for example, about 1,300,000 teachers in the United States, and it is to be fairly assumed that most of them are enumerated among our patients. Add to these their families, and the group should be about 3,000,000. These have been reached, with the possibility of spreading mouth hygiene information by and through them. There are also the other professional, and business groups.

In addition to these, the clientele is steadily being increased through educational efforts, many of whom are unable to pay for adequate service, resulting in the establishing of innumerable clinics, in schools, philanthropic organizations, hospitals, etc. These give service to the poor, the private practitioner to the rich, while the middle man is ground in between the two.

The Academy basically stands for prevention, as does all medical science. How does it live up to this obligation? Perhaps research will give the answer, for tooth cleansing by the patient and prophylaxis by the dentist are only a part of the program. Such experiments along dietary lines as are being conducted through the University of Michigan Dental School, the Forsythe Infirmary, Eastman Clinic, Guggenheim Foundation, etc., seem to point the way. The Basic Science Laboratory of the University of Cincinnati presents certain premises as to the irradiation of foods that give promise of better results in the control of caries and periodontal disease, although their experiments were not conducted specifically to prove these points.

The American Academy of Periodontology accomplished a most excellent work when it published "Dental Education Publicity" written and compiled by a Committee: C. B. Fowlkes, Chairman, Gillette Hayden, C. H. Schott and A. H. Merritt. This is the only general educational material of the organization, one of whose cardinal principles is prevention.

[1]Read before the American Academy of Periodontology, Nineteenth Annual Session, Buffalo, N. Y., September 8, 1932.

The present economic depression has brought to a focus the realization of the medical profession (which includes the dental) as to just what their attitude to the public at large is, and it behooves us to carefully analyze and study the situation.

Various propositions have been presented in an attempt to arrive at some satisfactory solution. It is not generally realized how far we have gone in foisting upon the State many activities. The public school system has been accepted for so many years, that one hardly thinks of it in any other terms than as a Governmental function.

In Ohio, for example, we have Workmen's Compensation. This makes compulsory the payment of certain benefits to people injured in industry, and every employer must carry this insurance. Strange to relate, the business concerns are well satisfied with it, for it costs less than other insurance, and the employee is better protected. The fees allowed to physicians and dentists by this Commission are comparatively small. While it is not uncommon to hear the professions object, still they accept the remuneration.

At this point, excerpts in favor of State medicine are presented from a pamphlet compiled by J. F. Baldwin, M. D., Columbus, Ohio: "Public health is the foundation upon which rests the happiness of the people and the welfare of the nation. The care of the public health is the first duty of the Statesman."—Lord Beaconfield.

"The socialization of medicine has begun, and prevention and public health will be the new keynotes." *Editorial, New England Journal of Medicine, March 31, 1932.*

As to the assured advent of State Medicine, I can only affirm what I have repeatedly said, that I believe its coming is inevitable and that it behooves us as members of the profession to try to direct its course in such a way as to produce the best possible results for both the public and the profession. To oppose it I believe is absolutely futile and may interfere with our influence in directing it into proper channels. The Sheppard-Towner Bill became a law in spite of the well united opposition of the medical profession. If the leaders of the profession had co-operated with the advocates of the law, is it not probable that a law would have been enacted that not only would have been acceptable to physicians, but also would have been more beneficial to patients?

Easy-going optimism may ignore or minimize the debacle of the profession from the high ideal of service to the low standard of commercialism, but we may be sure that the struggle by the public for self protection will go on against professional greed until the desired object is secured.

In this discussion, by the term "State Medicine" is meant that form of the practice of medicine by which physicians are in the employ of the State, and each physician is, in a sense, a health officer. His professional income is entirely by salary, so that he has no worry about collections, and he has no financial interest

in the illnesses of his patients, or in advising or executing operative procedures. His interests, in other words, are identical with the interests of his patients, and the prevention of disease is, therefore, more important to him and his patients than is its treatment.

Two or three years ago, President A. E. Morgan, of Antioch College, addressed a meeting of physicians at which a paper had been read antagonizing anything like State Medicine. President Morgan, in his brief remarks, called attention to the struggle which had been made many years ago to install our public school system, and he said he hoped to live to see the day when the poorest child in a community could get as good medical service as that child is now able to get in the way of educational service. His comparison seemed to be most appropriate, and his argument unanswerable. The childless now pay their part for the education of their neighbor's children; why should they not pay for the health of these children? If education is a national asset as all believe, why is not health also a national asset? Free education does not pauperize those who receive it, neither would free medical service pauperize its recipients.

Medicine, corresponding to our public system of education, and, like it, not necessarily including the wealthy "Private group practice," is almost invariably found to be a purely commercial proposition; it certainly makes no provision whatever for the young man about to enter practice.

"God Keep Us From Free Schools."

Gov. Berkley, of Virginia, in 1675: "I thank God that we have no free schools nor printing presses, and I hope that we shall not have any for a hundred years, for learning has brought disobedience, and heresy, and sects into the world, and printing has divulged them, and libelled governments. God keep us from both." *(Robert Baird—The Christian Retrospect and Register—1851.)*

In view of the eagerness with which these public and salaried positions are sought by physicians, it seems evident that, so far as the rank and file of the profession are concerned, such positions are looked upon as desirable. The opposition seems to come, so far as it is organized, from those physicians, (chiefly so-called "specialists"), who are in a position, or expect to be in a position, in which they can charge large fees for mediocre professional services, so that their incomes are out of all proportions to the incomes of ordinary physicians, and of other citizens of equal intelligence and ability, who have expended an equal or corresponding amount of time and money for their education. This comparison applies particularly to professors in our colleges, clergymen, judges and other salaried men.

At the time when the cure of disease was the only function of the medical profession, payment for each particular service seemed the natural procedure, but now that the predominating function is the prevention of disease, the public is benefited, whose duty it is to provide suitable compensation.

The large fees charged by specialists have resulted in an over-crowding of the specialties, with still further increase in fees, and a corresponding decrease of

general practitioners. The competition between these specialists to secure "business" has resulted in the shameful practice of fee-spliting, the result of which is that a large number of unnecessary operations are performed, many by thoroughly incompetent men; so that with advancing fees and lessened skill, the public is being ground between the upper and nether millstones.

In several European countries, in which State Medicine is practiced, no one can pose as a specialist without having passed a second examination showing that he is qualified. In this country no such evidence of qualification is required in any of our states, and apparently no effort has ever been put forth by the profession through the American Medical Association or the American College of Surgeons, to secure such protection for the public.

The thinking men in the profession are coming more and more to feel that it is socially desirable and imperative that all the people shall receive the benefits of modern medicine.

Dr. Warbasse predicts community centers in which problems of the prevention of disease will be worked out, so that prevention would supplement the old idea of treatment. Society is going to demand the talents of the physician to that end.

"No man, no profession, no science, can afford to barter with a principle. Making friends with the mammon of iniquity may be pursued beyond the bounds of expediency into the fields of positive destruction." *(Editorial, Illinois Medical Journal, Jan. 1928.)*

It is high time that we study the problems of adequate service, so that the leadership shall come from well informed professional minds, and not from those interested in their own pecuniary reward. High ideals must prevail.

The start of the organized mouth hygiene movement dates back twenty-five years. Up to that time, dentistry was concerned almost entirely with operative procedures, a natural position, for we had been an organized profession only since the establishment of the first dental college, then seventy-five years old. Naturally when the Oral Hygiene movement was launched, its first development was along empirical lines. Many at this meeting will recall how we started without slides, placards or paraphernalia of any kind. Now we have a well organized movement; quite a number of dentists are devoting their entire time to the work, through whom a large part of the development has come. In order to prove that this work was essential to educational progress in the early days, much was demonstrated through the so-called *experimental dental class.*

It is an interesting situation that the only comparisons made upon children whose mouths were changed from an unhealthy to a healthy condition should have taken place more than twenty years ago. Dr. Ebersole, one of the great pioneers, instituted a series of experiments at that time in the Cleveland Schools.

About forty children were selected with the worst possible mouth conditions. A series of tests prior to rendering dental treatment, were made. These

were physical, psychological, scholastic, attendance, behavior, appearance, manner, home environment, and any others that would have a bearing upon the case.

At the end of the school year, improvement announced was one hundred percent, two hundred percent, and even four hundred percent. This no doubt was true, but two vital factors were overlooked. Firstly, it was a serious mistake to select only the most exaggerated cases, and secondly, there should have been a control group.

Following these, in Cincinnati, we, believing that these experiments should have been made in an ordinary class room upon all classes of children, selected two classes in a school situated in one of the poorer districts of the city. They were of the same grade; the age, environment and general conditions of the children being equal. A series of psychological, physical, dental, scholastic, attendance, behavior, appearance, manner, social and other tests were made, and tabulated at the beginning of the school year. After this, the mouths of the children of the "experimental class" were all placed in a hygienic condition, those of "the control" being merely examined. The children of the "experimental class" were given regular toothbrush drills, periodical test dinners, at which they were taught to chew, and a general supervision of the mouths was regularly maintained. At the end of the year, all the tests were repeated, and a comparison made between the two classes. Mr. Hauer, the principal of this school, reports the following as the results in his summary:

Scholarship

"In the latter part of January, an examination was held, and both classes were given the same questions. The teachers were conferred with as to how each question should be marked, and where there were several parts to a question, as to the value of each part, so that all the pupils would be marked alike. I afterward looked over the papers to see how the pupils did, and found that they were marked in accordance with instructions. The class average in arithmetic, geography and spelling, the subjects that would test their scholarship, was 16.5 percent higher in the Experimental Class than in the Control. In other words, the class average in these subjects was 85.4 percent in one class and 68.9 percent in the other. The lower average of the two was, I thought, about normal, because the questions were not easy. The difference may not be wholly due to the dental attention given to the children, for one thing entered into the experiment which we at the school could not control, (though we tried to)—namely, that the Experimental Class discovered at the end of two months that it was such. I mention this fact in order to treat this topic fairly and impartially.

"Physical Improvement—The general appearance of the pupils, for a downtown school, had been above par. In some instances the improvement in this direction could not be appreciated unless one knew the pupils before and after the experiment. It is a pleasure to note the pride they had in their personal appear-

ance, and the splendid manner, spirit and general attitude they manifested in their class work."

Miss Conkey, who had charge of the psychological tests, says in her conclusions:

"In a general summary, we find that there is a decided lead taken by the Experimental Class. This holds true in all but one of the psychological tests. In the purely physical test the lack of gain in weight might justly be accounted for by the strain and nervous tension incident to the dental treatment, which in some cases continued over a long period. It is quite possible also that the gain in the various tests would have been greater had it not been for this conflicting element.

"While the Control Class led six times when the tests were first given, it led but once in the final series. On the other hand, the Experimental Class maintains her own four leads and passes five of the six leads of the Control Class."

The results, however, are plainly seen and the children enter into the work with an enthusiasm that is scarcely believable. It has been necessary this year to cleanse the mouths of but three children out of a total of fifty a second time, and it can be said that these three would be quite up to the average of the ordinary child applying for service in our offices.

A very effective dental organization can be built up for this work, which will assist when the time comes to raise funds to establish clinics. This is the way in which it was done in our town many years ago:

Lectures and Exhibits

There is no one single approach that has the value of the Mothers' Groups. Educate your dentists in the art of speaking before them. If sufficient time is allowed, the stereopticon may be employed. Great care should be taken that no slide is allowed to remain on the screen more than a short time. Models and placards are always of great assistance, and if a toothbrush drill by the children is presented, you can fairly see the mothers' faces beam.

At the present time, practically all the material for exhibits and lectures can be duplicated.

Dental Clinic

After the first educational seed has been sown, there will be a natural demand to have defects remedied. The private practitioner will soon discover that more children are coming to his office. Strange to relate, many of our confreres will not accept the responsibility of handling these cases, and much harm will be done by their stating that the work is not necessary at an early age. This, however, has been brought to public attention so often, that there is a move in the right direction, in addition to which the pedodontist has appeared upon the scene, and is very skilfully directing attention to the importance of children's work.

Except in a very small community, volunteer service in clinics has not appeared to be a success. The reason is that a practitioner will volunteer for a certain

definite time, and then allow a long interval to elapse before he returns. Meanwhile, some other practitioner will treat the case, with the result that little or nothing is achieved, and the volunteers lose interest.

A better procedure for the small community is to take the cases directly into the private office, finish the case, then take another one. Even better, is to establish clinics, with paid workers under adequate supervision. Great care should be taken if this is done not to lose the interest of the volunteer.

It is essential to keep the dentist in contact with the social phase of his work. There is but one effective way—make him a part of it.

Just as soon as he has assisted in starting a mouth hygiene campaign in his community, and it is turned over to a paid worker, the slogan "let him earn his pay" is adopted, upon which the volunteers work him to the limit, and they themselves automatically drop out.

The same attitude of mind is assumed by the layman. He must be aroused again and again as to the necessity of a healthy mouth—hence, the *educational campaign.*

Quite an extensive report of our recent campaign appears in the Journal of the American Dental Association for June, July, August, and September, 1932, for which reason the detail will be omitted.

It demonstrates how many agencies are essential to attempt an extensive educational effort. The Public Health Federation, as its name implies, is a group of delegates interested in any phase of health work, selected by their respective organizations.

Amongst the several sections are tuberculosis, cancer, child hygiene, mouth hygiene, mental hygiene, social hygiene, clinics and dispensaries, day nurseries, health education, heart council, housing and nursing.

All of these are headed by the Cincinnati Dental Society and the Public Service Dental Society, which operates free clinics, and is that part of the local dental society in charge of all social work of a dental character.

To these must be added practically every health activity, including the medical profession, Board of Health, Board of Education, and even some religious organizations.

The dentists, some months before, were instructed in the art of talking before various groups, and we are proud to say that at their first meeting, on a hot summer night, sixty-five were in attendance.

In evaluating this work, your essayist believes that the outstanding features were:

a—The coöperation of an unusual number of organizations;

b—The high percentage of dentists who assisted either with actual service or financial contributions, in most cases both;

c—Large amount of space of newspaper publicity;

d—Radio publicity;

e—Not one serious interference of any kind through almost an entire strenuous year.

Volunteer Service

Due to the times, a large number of former self-supporting individuals cannot avail themselves of dental service. Our clinical facilities are not adequate to take care of this problem. Volunteers were called for to take cases into their private offices, with the following result, and the effort is really just beginning to work effectively.

CINCINNATI VOLUNTEER SERVICE
August 25, 1932

132 dentists volunteered out of a possible 225, including specialists, some of whom were not equipped to do the work.

Applications on file	629
Active cases	196
Finished cases	146
Cancelled or withdrawn	31
Applications received	1,002
Fillings	1,092
Prophylaxis	159
Extractions	220
X-Rays, dentures, bridges, etc.	12
Number of hours	520
Value of service	$3,276.00
Amount collected	66.70

Colored Dentists

Number of volunteers	13
Number of hours	12½
Finished cases	5
Active cases	13
Broken appointments	8
Total patients	26

Summer Round-Up

Children enter the Kindergarten department at from four to six years of age. Through the Mothers' Clubs, early in the spring, an effort is made to ascertain in each district who these will be.

Each home is visited in order to interest the mothers of these prospective pupils in a thorough physical check-up. At a stated time, physicians, nurses, and dentists, go over the children; they are instructed as to the procedure for having the children treated, so that when they start to school in the autumn, any defects will have been corrected. The following shows the 1931 results:

SUMMER ROUND-UP 1931—AGES 4 TO 6 YEARS

Children examined	3,119
Need no attention	580
Need attention	2,539
Perfect mouths (no fillings or defects)	420

No. defective teeth	15,800
No. permanent defective teeth	519
Deciduous molars missing	657
Abscessed teeth	746

Re-Examination in Fall

Children examined	2,538
Children not examined	581
Had dental work done	1,111

Floating Prophylaxis Clinic

After the free clinic service was established and the Community Chest allowed $10,000.00 per year, the Board of Education $36,000.00, it was realized that more preventive measures were necessary.

For more than five years, groups of dental hygienists, with assistants, traveled from school to school, making examinations, lecturing before classes and doing prophylatic work.

There is a follow-up to discover how many have taken heed. Buttons are given to each child when his dental defects have been corrected, and then special banners are presented to the class rooms that are one hundred percent.

The private practitioner receives many of these as pay cases. It is most interesting to note the desire of the children to be amongst the elect. In the early days of the activity, there was some antagonism from the dentists who thought that practice would be taken from them. Now they know it has had exactly the opposite effect. About the only complaint is that occasionally people who are not entitled to free service take advantage of it. These cases are always carefully investigated when reported.

As was to be expected, the advertising dentist has availed himself of our propaganda. We are now compelled to combat his pernicious activities.

At this point, the action of the American Dental Association in advocating newspaper advertising by ethical practitioner groups deserves some notice. *It is a step in the wrong direction.* Are we to adopt the standards of the unethical? Our advertising should consist in educating the people through schools, clubs, exhibits, etc., as to the value of dental service in a dignified manner, and not to insinuate that we are looking for business.

It appears to your essayist that it is merely an attempted easy way to reach the public. However, such a method will probably fall of its own weight, it being too expensive to effectively do paid newspaper publicity.

Some of the so-called economic courses for dentists have gone too far in an attempt to make him a money maker. They present many features that are worth while, but in the final analysis the impression is left that the practitioner, and not the patient, is the more important.

As suggested earlier in this paper, the American Academy of Periodontology should now interest itself in mass prevention, and also coöperate with existing organizations toward that end.

Symposium On Periodontal Diseases—A Résumé Of Present Day Knowledge[1]

I. Receding Gum Margins

By ANDREW J. MC DONAGH, L. D. S.
Toronto, Canada

BECAUSE of its apparently universal occurrence, one is apt to believe that as he gets older the gums necessarily recede and expose the roots of the teeth, but according to an article in the July, 1931 Journal of Periodontology, by Dr. Bernhard Gottlieb, the teeth are continuously erupting which would account for the apparent recession of the gums.

I am showing some slides indicating that both of these theories are open to question. There are facts which support the doctrine of continuous eruption; Dr. Agnew for instance has slides of specimens demonstrating the fact that epithelial nests are nearer to the apices of the teeth in some cases than others, presumably nearer the apex in young people than in older individuals. This would mean, that, other things being equal, the epithelial nests either travel away from the teeth, or the teeth travel away from the epithelial nests. His evidence did not record the habits of the individual in regard to the closing of the teeth when swallowing, and so forth, but he has records showing the age of the individuals, which were taken from those cases in which occlusion was possible.

One of the important factors which would have a very definite influence, would be the pressure exerted on the teeth during an entire day, principally in the act of swallowing. I have a slide showing the results of swallowing improperly in one particular case; a young lady's teeth were ground two years ago so that the anterior teeth came into contact but when she presented recently I found that the posterior teeth had elongated to such an extent that the anterior teeth did not come within 3mm. of contact. (Fig. 1.)

On investigation I discovered that she had the habit of swallowing without closing the teeth together. When this was brought to her attention she endeavored to rectify the fault and consequently brought pressure to bear on the back teeth when swallowing; in three weeks' time the space between the anterior teeth was reduced well over one mm.

This emphasizes the fact that lack of function will cause teeth to extrude from their sockets; I do not believe, however, that it proves continuous eruption in case of normal function.

Again I have seen cases in young people, where one tooth has received a blow, and that tooth has become ankylosed and all the other teeth around it have progressed in eruption, giving it the appearance of having receded. On the other

[1]Read before the American Academy of Periodontology, Nineteenth Annual Meeting, Buffalo, N. Y., September 9, 1932.

hand, I have transplanted, and re-planted a great many teeth in the adult mouth, and so far as I can remember I have not seen any with the appearance of having receded into the jaw, notwithstanding the fact that at times they were without pericemental attachment. It must be remembered, however, that most of these teeth do not last more than three to six years; though a few may last ten to twenty years or even longer.

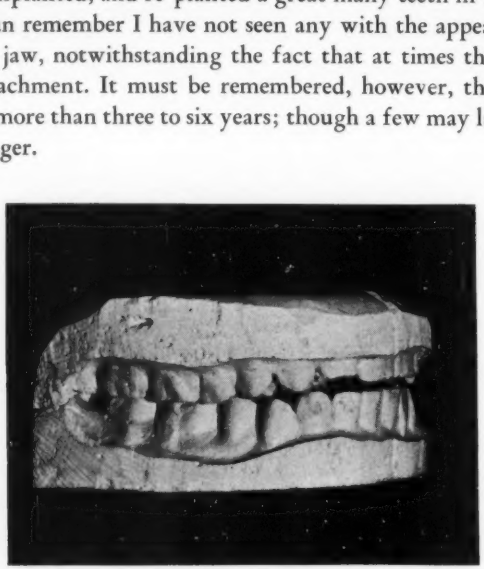

FIG. 1

Now the reason for paying attention to the doctrine of continuous eruption is that, if it be true, one might as well throw up his hands when he sees receded gums, since he will be unable to do anything for it. However, I hope to show that gum recession can be stopped, and that it is rarely, if ever, hopeless.

I shall show slides which demonstrate that with proper blood supply, including free circulation, the gums will hold their position in regard to the amount of the teeth covered.

If the alveolar process is broken down as shown in Fig. 2, and the patient is one whose food habits have not been normal, pockets which have formed usually leave the gingival tissues in practically their normal position, whether or not the pulps are vital. Fig. 3 shows the gum tissue as it appears in the mouth of this patient, at about thirty-four years of age, notwithstanding the fact that the hard tissues have been destroyed as shown in Fig. 2.

In this case, when looking into the mouth one would need to be very observant to discover anything wrong. The crown on the left upper central in this case, has been there for about twenty years, having been put on when the patient was about fourteen years of age. Please note that the gingival margin has not receded over the other teeth in the mouth much further than it has over the central which is crowned. The slight recession which has taken place is due to the fact that the circumference of the root has not been reduced at the gingival margin; in other words, the root was not bevelled when crowned.

This brings us to a side of the question which I have taken up before in former papers, namely, the fact that there is no recession of the gum margins over teeth that are properly crowned, no matter what kind of crown is used. Apparently the only requirement is, that the root be prepared as though it were to be crowned by a full banded crown.

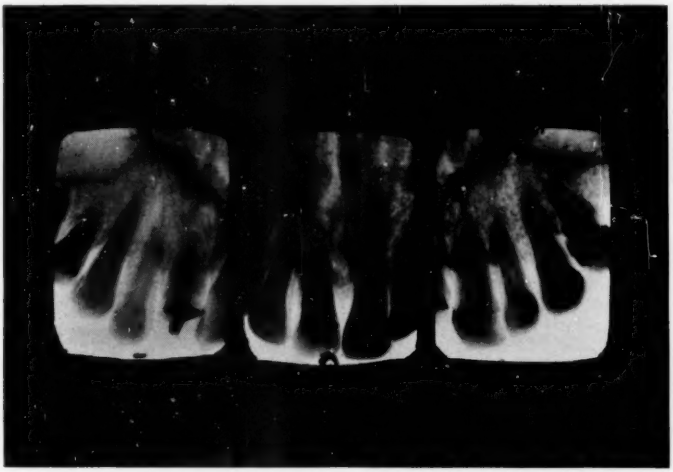

FIG. 2

FIG. 3

Fig. 4 shows a porcelain crown on the tooth of a patient, thirty years old. This has been on for seven years; all the other teeth in the mouth except this, show recession.

Fig. 5. Patient 48 years old, lateral crowned for 15 years; recession on all the teeth, except the crowned tooth.

Fig. 6 shows an upper central crowned for five years with a full band, the only one in the mouth with no recession. This patient was 15 years old when the tooth was crowned.

FIG. 4

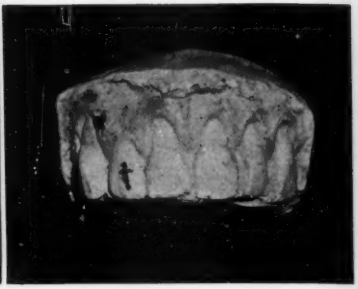

FIG. 5

FIG. 6

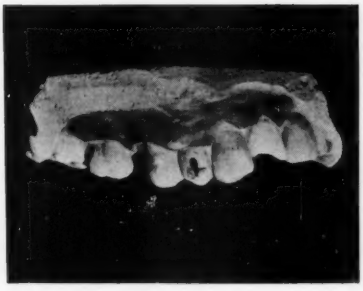

FIG. 7

Fig. 7. Patient 38 years old. An upper first bicuspid, shell crown, no recession. Recession on all the other teeth. Crown on tooth 14 years.

Fig. 8. Patient 35 years old. An upper lateral crowned 12 years. Same condition showing. Root in this case not banded.

Fig. 9 shows four crowns put on by Dr. John Ross of Toronto, 15 years ago; there is no recession. The remarkable fact about this case is that these teeth were protruding at the time they were cut off and crowned. Although one would expect recession to take place on such teeth, crowning them properly, evidently prevented it.

Dr. Hopewell-Smith claims that every adult, after the bony structure of the alveolus has become formed will have an immediate breaking down of that structure, resulting in a slight recession which he believes is normal because it is universal. That statement, I believe, is open to question. However, there is no doubt that halisteresis of the alveolar crest generally takes place, if prophylaxis is not practiced.

FIG. 8

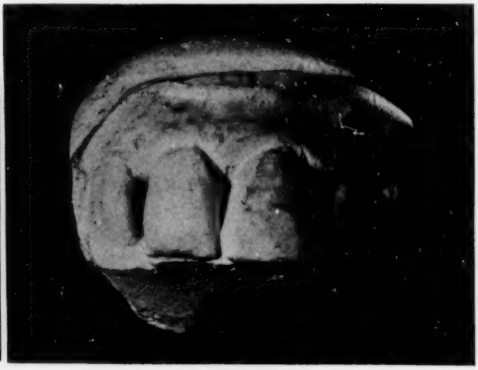

FIG. 9

To show that there will be no recession of the gingival margin if there is a free flow of blood, I want to call attention to some facts brought out in orthodontic treatment. I believe that Dr. George Grieve of Toronto, has done a great thing for orthodontia in teaching that the teeth are in abnormal position when they have moved forward from their apical base. He has also demonstrated the assistance orthodontic treatment can be to the periodontist; for when teeth have moved forward from their apical base they create conditions which to some extent, cut off the blood supply at the gingival margin. This is one of the greatest etiologic factors in the recession of the gingival margins, especially on the labial side.

The next slide is one which shows the work of Dr. Grieve. You will note that if the teeth are brought back to what Dr. Grieve calls their base, the gingival margin does not recede. However, if receded, it recovers its normal position when the strain on the underlying tissues is relieved.

Figs. 10a, 10b, 10c and 10d show the teeth of a child about ten years of age. This case was finished, as shown in Figs. 10b and 10c. The result, so far as the teeth are concerned, is good, and the appearance greatly improved, but, observe the recession at the gingival margin of the lower teeth. This case was regulated in the ordinary way by bringing the teeth forward.

Now take another case: Figs. 11a, 11b, and their enlargements 11c and 11d. Fig. 11a illustrates model of the teeth when this boy was about nine years old, but the teeth were regulated as shown in Figs. 11b and 11c. Note that, when the case was first started, the left lower central was showing signs of recession, but when it was finished, not only has no recession taken place on the other teeth, but the left lower central has had the gingival margin restored to nearly its normal position.

I am not showing the next slides with the idea of recommending the operation I am about to describe, because it is so seldom successful that I do not consider it a conservative operation. It shows, however, what can be done to restore gum tissue that has been lost. Fig. 12 shows the gum line receded to three-

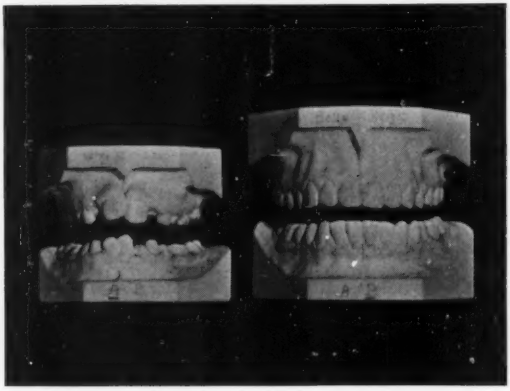

FIG. 10a FIG. 10b

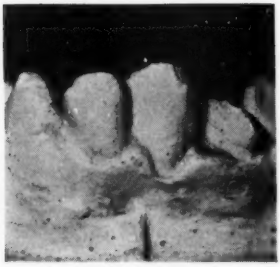

FIG. 10c FIG. 10d

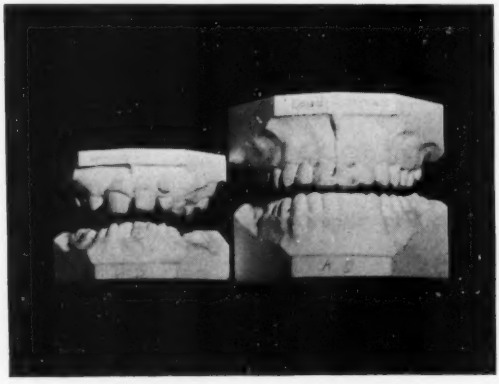

FIG. 11a FIG. 11b

quarters of the length of the root. Fig. 13 shows this same patient with the gum line being restored to normal. Fig. 14, the same tooth and gum in place six years after being operated on. Fig. 15 shows another case with the gum

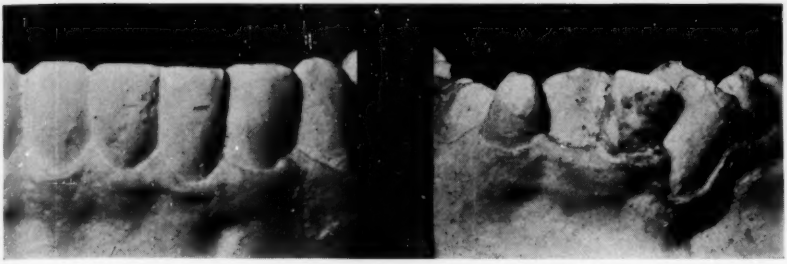

FIG. 11c FIG. 11d

line receded to practically the apex of the root. Figure 16 shows it restored to normal. This picture was taken some time after the operation was performed; I have no record of how long afterwards.

To sum up, there is one factor at the bottom of all recession of the gingival margin of which we have spoken, namely, the lack of a free blood circulation. It is not due to the loss of the alveolar process, or a vitiation of the blood supply, as has been suggested, or to the continuous eruption of the teeth, which no doubt would take place if there were not proper contact or proper use made of the teeth.

So far in this paper no mention has been made of the effect of dentifrices, mouth washes and stiff brushes. Nor has anything been said about salivary calculus as a cause, all of which have some effect in causing ulatrophia. There can

FIG. 12

FIG. 13

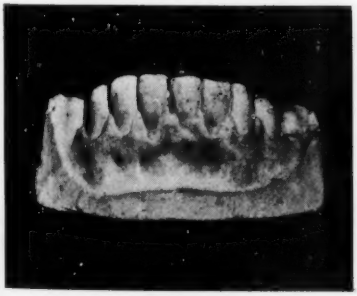

FIG. 14

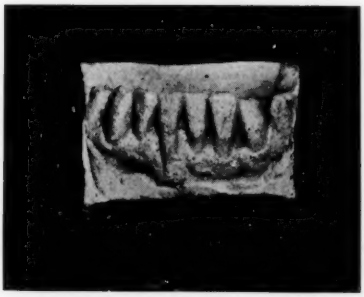

FIG. 15

be no doubt that brushing, if the conditions are favorable, may be responsible for some destruction of the gingival margin. Dentifrices also, and highly astringent mouth washes may, and probably do hasten gingival recession. Salivary calculus is another common cause because of its irritating effect; traumagenic occlusion may also be a causative factor. Nevertheless if the blood supply at the gingival margin is adequate it will be difficult for any of these extraneous factors to have much effect.

FIG. 16

Considering the evidence as shown in the slides we conclude that the breaking down of the alveolus as shown in Fig. 2 is not the etiologic factor in gingival recession. Fig. 4 to 9 demonstrate that recession of the gingival margins is the result of ulatrophia, due to a lack of free blood supply in the gingival tissues. The other slides seem to indicate that the lack of free flow of blood in orthodontic cases can, by proper treatment, be overcome; therefore, the etiologic factor is the same as in the other cases.

There are other methods than these for preventing gingival recession and for restoring the lost tissue to a certain extent but treatment is not within the province of this paper.

I have, throughout spoken of *receding gingival margins* not receding gums, or receding gingivae, because there is practically no such thing as receding gums. The condition represents an atrophy of the gingivae at the margin, consequently ulatrophia, which results in recession of the gingival margins.

II. Vincent's Infection

By CLAYTON H. GRACEY, D. D. S.
Detroit, Michigan

Current dental literature is, unfortunately, intermingled with much contradictory material on this subject. One positive conclusion, however, is possible, namely, that an infectious malady of a certain type is rampant and that its chief habitat is the mouth.

For the latter reason, dentistry, practically alone, has fathered this refractory disease, and is having its first opportunity to present, in the literature, the clinical and research findings of a purely infectious disease.

The first evidence of confusion, regarding Vincent's infection, appears in the group of terms under which it has been reported. Over thirty different names are recorded, each descriptive of characteristics, environment, symptoms, or etiology of this pathological entity. However, thirty-seven out of fifty more recent articles were titled, *Vincent's Infection,* and for the present, this seems sufficiently descriptive to warrant its continued use.

An increasing incidence of Vincent's infection, particularly that of the so-called chronic or subacute type, is conceded by practically all writers. Through the agency of the United States Public Health Service, the dental departments of most Universities and through the medium of questionnaires to private practitioners it has been definitely agreed upon that Vincent's infection is increasing.

Coincidently, it is proven that this infection is to a degree of an epidemic and endemic type and its graph has, to some extent, been charted and anticipated with considerable accuracy.

With the exception of a few writers, the etiology of Vincent's infection is, generally agreed upon. This is based on the theory of a symbiosis of the specific organisms *Bacillus fusiformis* and the *Spirillum* of *'Vincent-Plaut.'* Their constant presence in typical lesions has led to a general acceptance of the belief that they are the primary causative factors in the disease. The fact that, when the lesions are treated and the symptoms subside, these bacteria likewise become less in number and practically disappear, bears out the soundness of this hypothesis.

Another theory is, that the two dominant organisms are but developmental phases of a parent organism with varying degrees of virulence in different stages of mutation.

Still another qualifying suggestion is that a soluble toxic product of these organisms is responsible for the pathological changes rather than the organisms themselves.

The description and reported characteristics of these bacteria with their staining properties is quite consistent. They are definitely anaerobic and saprophytic. Due to the concomitant presence, in the lesions, of aerobic organisms it is the opinion of some that the oxygen in the regional tissues is consumed, leaving a more suitable anaerobic field for the growth of the fuso-spirochetal group.

The importance of the factor of lowered resistance is recognized by all, and this phase of etiology is being more and more emphasized. Typical of this trend is the following quotation: "These organisms are opportunists and even the more virulent strains are harmless until the barrier of tissue resistance has been destroyed" (1).

Probably the least divergence of opinion will be found on the subject of diagnosis. The subjective and objective symptoms described, are so uniformly characteristic of this disease that there is but little left for question. These include all the cardinal symptoms of inflammation; recession and a punched out appearance of the interproximal crests; deposition of a grayish-yellow pasty slough with an irregular line of demarcation, associated with hemorrhage; salivation; throat irritations; tenderness in the tongue and faucial regions; swollen glands; increased mobility of teeth; characteristic odor; malaise; increased temperature; accelerated pulse rate; pallor of skin; mental depression; insomnia; lack of appetite.

However dependable such a combination of any or all the above signs and symptoms might be, it is emphatically asserted that diagnosis cannot be complete without the aid of the microscope. "In order to make a positive diagnosis of Vincent's infection it is necessary that the clinical diagnosis of the disease be substantiated by the microscopic examination. This is not a generality but a hard and fast rule" (2). The practicability of this statement is questioned by few scientific minds. Encouragement in the use and application of the microscope in diagnosis is recommended and its more general use is earnestly implored.

Reports of but few microscopic studies of tissue from Vincent's lesions are found in dental literature. One author presents, in his characteristically scientific and lucid style, an interesting and detailed description of the histo-pathology of this lesion. This treatise, prepared by Harold Keith Box, is titled "Necrotic Gingivitis," and has been presented, serially, in recent numbers of a dental magazine. In it are explained the inflammatory processes with the association of necrosis and desquamation of superficial epithelium. These are described as accompanied by a marked inflammatory exudate, largely of fibrin, which in turn incorporates the cells of the exudate as well as the necrotic cells of the antecedent epithelium (3). A vivid picture of the formation of the characteristic slough of Vincent's is portrayed.

Much contradiction is to be found regarding treatment. To report even a small percentage of the various medications and the technique of their administration, would be entirely out of place.

One cannot but be concerned by the number and variety of chemicals offered for treatment. Acids of a most corrosive and coagulative type are included in the list, with conclusive evidence presented with each that through their use the virulence of the organisms in Vincent's infection may be curbed. Too little, however, is said of the undesirable effects on tooth and soft tissues which may follow the indiscriminate use of some of the drugs recommended. Even the slight etching of enamel and dentine caused by some of the acids suggested must be considered adverse to dental health thru its potential influence on hygiene and caries.

In view of these facts the following requisites for an efficacious remedy in the treatment of Vincent's infection are worthy of quotation.

(1) "The remedy employed should be absorbed to a greater or less extent by the tissue.

(2) "It should be antiseptically active for an appreciable time.

(3) "It should have a solvent action upon the necrotic tissue characteristic of the lesion.

(4) "Its antiseptic action should be specific upon one or other components of the symbiosis, for if one of these is eliminated the symbiosis is destroyed" (4).

The anaerobic property of the organisms of Vincent's disease is, of course, responsible for the recommendations of oxidizing agents either as the primary medication or as the supplementary treatment, through the agency of mouth washes. Their therapeutic efficacy over other drugs is sometimes questioned but their milder properties would seem to commend their application, in most cases.

The detergent property of hydrogen peroxide and chromic acid, the most widely accepted oxygen producing agents, is credited with some of the potency of these drugs, in their action on Vincent's disease.

Besides the oxygen liberating drugs, there are offered groups of the arsenical compounds and other specifics for the spirochetes. Most worthy of these are neo-arsphenamine and sulpharsphenamine. Neo-arsphenamine in powder form, packed around the necks of all the teeth in the mouth, is found, by some clinicians, to be more advantageous than the glycerine solutions because it forms a sticky mass, which is retained much longer in contact with the tissues.

Numerous bactericides of varying strength and potency are to be found in the lists of suggested treatments.

Of interest to the dentist, from an operating standpoint, are the various topical application techniques recommended in conjunction with certain medicaments. Some require a thorough removal of all mouth debris and accumulations of slough and filth before the application of the drug. Others permit of the flowing of chemicals over the ulcerous conglomerate.

One of the most effective treatments of this latter type is recommended by Dr. J. F. Adams of Toronto. This consists of the application of "Churchills Tincture of Iodine," followed immediately by a 35 % aqueous solution of silver nitrate.

Drying the tissues is not even recommended by the advocate of this technique. "The chemical reaction between the two solutions results in the precipitation of insoluble iodide of silver in the necrotic tissue which within thirty-six hours is cast off leaving a healthy epithelial surface" (5).

Seldom, in the literature, is there found allusion to a factor which is important in treatment—that of 'acquired organism resistance' to medicinal therapy.

The action of a drug, on repeated applications, is oftimes decreased. Alternating the medication on successive treatments is considered, by many, to be advisable.

Unanimity, in more current writings, is found on the question of early prophylaxis. Many writers still hold to the precept that all instrumentation and kindred operative procedure should be omitted in early treatment. There seems to be but little basis for this theory as has been proven clinically by numerous operators. "The establishment of good hygiene at the earliest possible moment consistent with the patient's comfort is clearly one of the first principles in the treatment of Vincent's infection. In making this statement it is realized that there are those who still cling to the older conception of drugs first and cleanliness afterwards, a mode of procedure that explains in many instances the long drawn out treatment so often reported in Vincent cases" (6).

The status of the toothbrush, in Vincent's treatment, is seldom commented upon. Its judicious but vigorous use and the need for discarding the old brush, as soon as the acute phase of the disease has subsided, is advocated.

Specific dietary recommendations follow the rule that foods be generally of an alkaline reaction and of such character that elimination be stimulated. Smoking and excessive use of alcohol are contraindicated, from a local as well as from a general viewpoint.

Intra-venous or intra-muscular injections are not in favor with most writers. But few tangible results have been demonstrated and in some cases even, it is reported to apparently aggravate the local condition.

Vaccine therapy is condemned as ineffective, having to be made up from secondary organisms present in the lesions, as it cannot be made from the Vincent organisms direct.

The Ultra-violet ray is considered useful only as supplementary treatment. Systemic irradiation is often advised, coincident with local application.

If there is a systemic condition present which has predisposed the tissues to infection or which is concurrent with the disease, general as well as local therapy is regarded as essential.

Numerous records, in the literature, emphasize the possibility of systemic involvement from acute Vincent's. Continuity of tissue, aspiration, the blood stream and the lymphatic system, combine to make probable the reports that, incidental infections, such as lesions of the respiratory passages, brain abscesses, pulmonary gangrene, and others are directly caused by the focus of a Vincent's infection in the mouth (7).

Prognosis is regarded as favorable when the condition is properly diagnosed and treated, and when one has the coöperation of the patient. An important factor in prognosis is the possibility of primary lesions such as: tuberculosis, diphtheria, herpes, leukoplakia, pemphigus, mercurial stomatitis, and other diseases of oral manifestation, being overlooked during the course of a Vincent's lesion of

a secondary nature. In all such cases, it is desirable that the elimination of the Vincent's infection should be promptly carried out because of its unfavorable influence on other conditions and also to prevent its possible extension and destruction of tissue.

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III. Etiology of Periodontoclasia

By A. W. BRYAN, D. D. S., F. A. C. D.
Iowa City, Iowa

Although the symposium title refers to present day knowledge, it may not be amiss to briefly enumerate some of the theories of causes advanced in the early periods of dentistry, leading to the accepted beliefs today. The main issues as to etiologic factors are immediately brought to mind when we mention "systemic factors" and "local factors." For many years the relative importance of these two groups caused much debate, there being many enthusiastic supporters for both.

For years an effort has been made by investigators to find one specific factor as the cause, as is indicated in the list of theories which follow.

Among the rather vague suggestions that have been advanced is one called "precocious senility." This was based on the observation that certain individuals develop "early old age" and, therefore, the supporting structures of the teeth are affected by degenerative changes and are eventually lost. The proponents of this theory pointed to the occurrence of grey hair in the young as another symptom of this early senility.

Another group believed that as individuals advanced in years the blood supply to the supporting structures decreased, and thus the integrity of these

tissues was affected. Certain aspects of this theory fit in with some of the present accepted beliefs, as indicated in the emphasis placed upon proper massage and stimulation to the supporting tissues to insure good blood supply. But the matter of age was not the important factor.

Another theory advanced was that the alveolar structures are of a transitory nature, as evidenced by their rapid absorption around the socket of a tooth after extraction. This claim carried little weight because any osseous structure is subject to quick change when placed under pressure, or when its nutrition is changed. The rich blood supply about the alveolar plates would, however, have some influence on the rapid tearing down of unneeded portions when a tooth is lost. Likewise, this same generous blood supply should benefit the tissues in a diseased condition, for it can carry much nutrition to, and the products of infection away from, the tissues when local causes are corrected.

A theory which all who are present will recall was the so-called amoeba theory. Two groups of investigators almost simultaneously reported that they were able to demonstrate amoeba in practically all cases of periodontoclasia, claiming that they were the specific cause. This theory gained many supporters, and for a time the sale of emetine and ipecac increased by leaps and bounds, and many were the sore arms resulting from the use of the hypodermic. As we all know, very little importance is attached to the presence of amoeba at this time.

The amoeba is not the only organism that has been held to be responsible. Always some one has been searching for some specific type of infection as the cause of dental periclasia. Infection no doubt plays an important role and practically every student of periodontia today recognizes it as a real factor to be considered. Some members of this Academy consider it of major importance, and we would not minimize its part in etiology.

Another factor, considered of much importance at present, is that of irritation. Under this term we think of the chemical, bacterial, and mechanical phases. One particular type of the mechanical phase has come into much prominence and has been the subject of much debate,—namely, traumatic occlusion.

Present tendencies are to group the etiologic factors into three main divisions, as indicated in the chapter on Etiology of Periodontoclasia in "Periodontal Diseases" by Merritt. We have first those of systemic origin; second, those of local origin; and third, interrelated factors from the two foregoing groups. Out of long years of study and observations by periodontists and pathologists have come results which lead the majority of us to believe that, in most cases at least, a vicious cycle is involved in which local conditions affect systemic health. These in turn affect local resistance, and, therefore, making it necessary that both groups be considered.

In considering causes it is convenient to divide factors into primary and secondary, classified in some of the older texts as active and predisposing. It is generally accepted by the profession that irritations are the main primary etiologic

factors. As stated, these irritations may be chemical, bacterial, or mechanical. Under bacterial may be considered the various types of infections that have the power to destroy tissue or lower its resistance, and which should be given consideration in diagnosis and treatment. The factor of chemical irritation is directly related to bacterial because of the part which infection plays in the production of irritating acids and the formation of toxic by-products of bacterial growth.

In the matter of mechanical irritation are several factors to be considered. As presented in Stillman and McCall's Etiological Chart, we should consider those factors which have to do with injury to the marginal tissues, and those also having to do with injury to the deeper periodontal tissues.

In marginal irritation we have calculus, sordes, etched tooth surfaces, ill-fitting bands, etc. which are associated in one way or another with nearly every case of gingivitis. If we can conceive of a case being caused by local factors only, here is the place where such theory would be most logical.

In considering mechanical irritations affecting the deeper structures we encounter that much discussed factor, traumatic occlusion. I believe that much of the debate on the importance of this factor has been due to a misunderstanding of the term. Those who place little value on its importance say, "teeth were made to masticate food and the tissues will stand the stresses necessary for that use." This premise is granted, but we must remember also that nature built the supporting tissues to withstand stresses in certain directions, and when the stress in another direction becomes excessive the supporting tissues suffer. I can see no difference between alveolar destruction during excessive stress (traumatic occlusion) and destruction during orthodontic movement, except that in one the force is uncontrolled and intermittent, while in the other it is controlled and positive. Absorption takes place in both cases, but in controlled pressure nature is able to build bone behind the moving tooth. Much of our misunderstanding has been in believing that the tooth should stand stress in *any* direction. The abutments of river bridges will stand tons of load when that load is directed in line with the long axis of the abutment, but the abutment support gives way, and many bridges are washed out by high water and debris exerting excessive *lateral* stress. Traumatic occlusion to me means excessive lateral stress, and must be considered as a factor of importance.

Stress on teeth is a relative term, and must always be considered in its relation to the integrity of the supporting tissues. At this point some of the systemic factors enter the picture. If we could conceive of two individuals with the same type of occlusion and the same muscular strength, and therefore exerting the same amount of stress,—in one the stress could be excessive and in the other normal. The difference is in the integrity and the adequacy of the supporting tissues. A systemic condition at any time may change the fulcrum point between stress and resistence, and thus change the relative stress.

Closely associated with health of the supporting tissues is the factor of diet.

In view of the results of the researches of Howe, McCollum, Grieves, and others, we must concede that what a person takes into his body as food, has a marked influence upon the quality of the tissues. There is much difference of opinion as to the place diet should occupy in the etiologic chart, but we must admit that our body tissues are "what we eat." Food builds our tissues;—food must maintain them.

Any systemic disease that affects metabolism in such a way that the normal processes of growth and repair are interfered with may be a factor in the production of any type of periodontoclasia. It is generally believed that the anemias, tuberculosis, and diabetes have a rather definite effect upon the integrity and resistance of the alveolar tissues. Any general disease that lowers general body resistance cannot help but influence the health of the oral tissues.

Pregnancy is a factor which must be considered. It is a matter of common observation that women whose oral conditions have been excellent before pregnancy, begin to suffer from noticeable loss of alveolar bone when that condition exists. Pregnancy being a normal physiologic process, it is questionable whether it would not be better to lay the blame at the door of inadequate diet during a period of increased mineral need.

Another factor which should be considered is that of the reactions of different individuals to the same irritant. We see two individuals who are seemingly comparable in age, size, health, etc. Both have large collections of salivary and serumal calculus, but one shows only a moderately severe gingivitis while the other shows a marked amount of loss in the supporting tissues. They have different reactions to the same irritation, and, while this condition cannot be classified as an etiologic factor it must be considered as the difference in resistance to many local causes.

To summarize the important points it may be said that the consensus of opinion as expressed in modern literature on the subject of etiology of periodontoclasia is first, that factors of local irritation such as calculus, placques, bacteria, chemicals, etc., may be logically assigned as the cause of most types of gingivitis; second; that factors of irritation in the form of excessive stress are frequently the primary factor in the destruction of the deeper supporting tissues; and that when pockets are once opened, infection is a major item; third, that systemic factors are important in that they determine the ratio between resistance and cause, and complicate the case by the formation of the vicious cycle, in which both local and general factors are at work.

IV. Treatment of Periodontoclasia

By J. CLARENCE SALVAS, D. D. S.
Philadelphia, Pa.

Our greatest interest in the study of disease is its treatment. We are so absorbed in this phase of the subject that we are apt to become oblivious to its

etiology, and pathology, as we are also of its course and progress. To avoid empiricism and to place treatment on a scientific and rational basis it is necessary to have an intelligent comprehension of the etiology and pathology of disease. Research workers and the observations of those who have studied the course and progress of disease have made this knowledge available; there is no excuse therefore, for the dentist to adopt empirical methods of treatment, depending upon drugs and nostrums, which have no place in the hands of the intelligent clinician. The treatment of disease consists of methods which aim to restore the tissues to health by removing those causative factors concerned in the creation of the disease and in stimulation of the biologic forces of repair. It has therefore two objectives: first the correction of the pathologic conditions of the periodontium by operative methods, and second the elimination of the underlying factors of disease by operative and post-operative care, in order to restore and maintain the investing tissues in health.

Before attempting the treatment of periodontoclasia, it is important to have a clear understanding of its etiology. This is more or less complex, there being a number of factors entering into each case. There is one characteristic, however, which is common to all, namely: it is in the gingivae that the first symptoms of disease appear clinically. In all cases the important etiologic factors are bacteria, lowered tissue resistance, and unbalanced occlusion.

It is important as a preliminary to treatment that a complete radiographic examination be made, for in no other way is one able to ascertain the extent of alveolar atrophy about the involved teeth, the condition of the periapical tissues, pulpless teeth, etc. The periodontist will experience little difficulty in correcting conditions visible to the eye, but the underlying factors will tax his skill.

The early recognition of periodontal and perialveolar abnormalities is of the greatest importance as it enables the dentist to institute treatment of the disease, which if taken in its incipiency, can be easily arrested. The radiograph, therefore, is indispensable for diagnostic purposes, as well as a guide to treatment.

An important feature of the treatment of periodontoclasia is to impress the patient with the fact that to accomplish a full measure of success it is essential to have his earnest and persevering coöperation, for in no other way can one hope to control the bacteria which are such an important factor in its etiology. This means, and it must be made clear to the patient, that a high standard of mouth hygiene must be established and maintained. Furthermore, the periodontal tissues must be frequently stimulated in order that their resistance to bacterial infection be increased. By no other means can a cured case be kept in health. Carelessness in the personal care of the mouth inevitably results in reinfection regardless of other curative measures. This is the weak point in our treatment of periodontoclasia—weak, because we depend on the intelligent coöperation of the patient—something very difficult to have for any length of time.

There are no stereotyped methods for the treatment of periodontoclasia.

It must be modified to suit each individual case. In each case there must be a complete radiographic survey. This is followed by a cleansing process. My method is to swab the mouth with large rolls of cotton saturated with pure hydrogen dioxide, paying particular attention to the interdental tissues. The mouth is then given a prophylactic treatment, after which a study is made of the occlusal relationships, using study models for this purpose. At this time the vitality of all doubtful teeth is tested.

In the effort to remove all sources of infection a comprehensive examination of all fillings, contact relations, crowns, bridge and plate attachments is made. It is imperative that proximal contacts be properly restored for there is no greater source of trauma of the septal gingivae than that produced by food impaction. Poor fillings, with broken down cervical margins, overhanging edges, ill-fitting crowns, both gold and porcelain, are sources of irritation and infection and must be corrected before one can hope to obtain permanent results.

The first step in treatment is the correction of occlusal faults. Functional occlusion is so essential that I cannot pass this point without emphasizing the importance of detecting any uneven distribution of stress, for this is a factor which has an intimate bearing on the health of the teeth and the investing tissues.

After this, we are prepared for instrumentation, which plays such an important part in the treatment of this disease. Surgery of the root surface, the seat of calcific deposits, is achieved by curettement, which means the removal, not only of the hard deposits, but the equally important necrotic tissue. The curettement of a root surface requires considerable skill. A delicate sense of touch is essential for it enables the operator to see with the end of an instrument the nature of the substance with which he is in contact. To achieve the requisite skill requires persistence and diligence, which should be a part of every dentist's armamentarium. The purpose of treatment is to reduce the inflammation in the gingival tissues, stop the flow of pus and tighten the teeth. To do this the pocket must be eliminated. Many dentists are unable to understand why pockets do not close after the etiologic factors are removed. They fail to realize that the epithelium lining the pocket acts as a separating medium which prevents the connective tissue forming the attachment, from uniting with cementum surface. The problem, therefore, is to remove the epithelial tissue, which is most effectively accomplished with Dr. McCall's depilatory solution—sodium sulphide 150 grams, sodium carbonate 40 grams, aquae Q. D. ad. It seems to have a special affinity for epithelium and does not destroy the adjacent tissue. The technique of applying it is simple and there is no discomfort to the patient.

The patient must be made to understand that pyogenic micro-organisms grow on the surfaces of the teeth with remarkable rapidity and that it is absolutely necessary that the bacterial accumulations be removed twice each day. They must be taught how to manage the toothbrush so that in their efforts to keep the teeth and surrounding tissues free from bacterial activity, they exert

sufficient pressure and friction on the gums to reduce conjestion, stimulate the gingival circulation and improve tissue tone. By this method of friction and physical exercise of the gum the depth of the pockets is reduced sufficiently to allow the epithelium to extend to the bottom of the pocket.

In surgery of the root surface, (subgingival curettage), I prefer the curettes as the most efficient instruments. They are more delicately constructed so that in operating one feels a keener sense of touch than with instruments of heavier construction. One is working in a field entirely hidden from view, therefore, it is necessary to be able to feel the nature of the substance being worked upon. I use McCall's curettes Nos. 17 and 18, also Nos. 3 and 4, 13 and 14 of the Younger set. Where pockets are deep I like plane instruments such as Nos. 13, 17, 11, and 15 of the Towner set. We must not lose sight of the fact that the entire root surface involved by the pocket is septic and covered with necrotic matter, serumal deposits and bacteria that must be completely removed. Therefore, the field of operation following curettement, should be thoroughly irrigated with a normal salt solution.

When any of the surfaces of the teeth or exposed roots becomes sensitive to cold, they are dried, coated with iodine, cleaned with a porte-polisher and covered with Kryptex which is allowed to remain for several days or until the tooth is comfortable. The only other occasion I have for using a drug is when the interdental tissues are considerably congested, as in persistent gingivitis or when the orifice of a pocket is swollen. Copper sulphate is used in such cases and one or two applications is generally helpful.

Surgical treatment for the elimination of pus pockets has a place in the treatment of periodontoclasia. I seldom have occasion to use it, however, excepting in deep pockets about the molars where there is extensive loss of bone, confined principally to the interproximal spaces, and where a deep pocket is created by the unattached and impoverished septal gingivae. This method has one decided advantage, in that it renders the field of operation more accessible making it possible for the operator to see exactly what he is doing.

I repeat, by way of emphasis, that one cannot lay too much stress on the importance of post-operative care. The control of the etiologic factors, such as bacteria and low tissue tone is absolutely essential to maintain the periodontal tissues in health.

I look forward to our having a clearer understanding of the influence of food intake on the physiologic balance of the blood. What are the factors in low tissue tone, lowered resistance, etc., which make the tissues susceptible to bacterial activity? Is it alkalosis or acidosis? If so, what are the factors back of these conditions? We learn from blood chemistry that the blood is 80% alkaline and 20% acid, and we know that any alterations in this relationship causes reaction in the tissues. The controlling factor back of these elements is food.

For the past two years my treatment for advanced periodontoclasia and Vincent's infection has consisted in part of a rigid food arrangement—not diet—as the patient can eat anything he desires but in proper combinations, the principle being that carbohydrates and proteids are not to be combined. During this comparatively short period I have experienced really remarkable results.

One cannot but be impressed with the fact in surveying the field of Periodontology and studying the character of the diseases that break down and destroy the periodontal tissues, that there is much to be learned from research. While advanced cases of periodontoclasia yield to a certain extent to skillful treatment it is, nevertheless, not entirely satisfactory as one cannot restore destroyed tissue. This emphasizes the importance of prevention and early treatment.

Correlation of Symposium on Periodontal Diseases

By ARTHUR H. MERRITT, D. D. S., F. A. C. D., F. A. A. P.
New York City

Receding Gum Margins

THE ONE factor underlying all recession of the gingival margins is believed by Dr. McDonagh to be "lack of free blood circulation" in the gingivae. It is not due to a loss of the alveolar process nor to a vitiated blood supply as has been suggested, nor even to the continuous eruption of the teeth, though this latter phenomenon may cause recession about teeth not in occlusal contact. Improper tooth brushing, highly abrasive dentifrices, astringent mouth washes, etc., may be factors in producing gum recession, as may salivary calculus and traumatogenic occlusion. These, however, are not believed to be serious factors when the blood supply to the periodontal tissues is adequate. It is asserted that "gum recession" can be arrested and brought back to a certain extent and also that the condition is one that "is rarely ever hopeless."

Vincent's Infection

While deprecating the contradictions in terminology Dr. Gracey calls attention to the fact that Vincent's Infection is the term adopted by the committee on nomenclature of the American Dental Association as one that is sufficiently descriptive to warrant its continued use. It is a disease which he reports as being on the increase and at times epidemic in form. It is caused by the fuso-spirochetal organisms in symbiosis, though the fact is emphasized that "even the more virulent strains are harmless until the barrier of tissue resistance has been destroyed." The diagnostic symptoms are given as inflammation, recession of the septal gingivae, sloughing, hemorrhage, salivation, odor, malaise, temperature, etc. The use of the microscope in diagnosis is urged. In treatment it is recommended that the drug used should be one that will be absorbed into the

tissues in order to exercise its therapeutic influence in the tissues beneath the surface; that "it should be antiseptically active for an appreciable time"; that it should be neither caustic nor destructive and should have a solvent action on the necrotic tissue.

Attention is called to the use of a 30% emulsion of bismuth tartrate as a remedy possessing all these qualities: also to the rather general use of oxygen liberating compounds and the arsenicals. One of the most efficacious treatment's is said to be Churchill's tincture of iodine followed by a 35% aqueous solution of silver nitrate. The importance of early prophylaxis as supplemental to the use of drugs is also stressed. A diet alkaline in reaction is recommended. Smoking and the use of alcohol is contraindicated. Intra-venous injections and vaccine therapy are of little value. Ultraviolet radiation may be useful as a supplementary treatment, but is not a specific. Prognosis as a rule is favorable.

Etiology of Periodontoclasia

Dr. Bryan divides the etiologic factors in periodontoclasia into three groups as follows:

First: Those of systemic origin

Second: Those of local origin

Third: Those interrelated factors found in the foregoing groups.

These he enumerates in the following order:

(a) Infection, which he believes plays an important role especially "when pockets are once opened."

(b) Irritations which are divided into chemical, bacterial and mechanical. Under the latter, traumatic occlusion is considered at some length and defined as an "excessive lateral stress," which should "be considered in its relation to the integrity of the supporting tissues."

(c) Diet: which in the light of researches referred to is believed to have a marked influence upon the quality of the periodontal tissues.

(d) Systemic disease: such as tuberculosis, diabetes and the anemias: Indeed, any disease which lowers body resistance is believed to adversely influence the health of the periodontal tissues.

(e) Pregnancy, under conditions of inadequate diet at a "period of increased mineral need."

In a summary, calculus, placques, bacteria, chemicals, etc., are given "as the cause of most types of gingivitis": excessive stress is frequently the primary factor in the destruction of the deeper supporting tissues, and lastly, systemic factors "in that they determine the ratio between resistance and cause" and "complicate the case by the formation of a vicious cycle."

Treatment of Periodontoclasia

The treatment of periodontoclasia has according to Dr. Salvas two objec-

tives, namely, the correction of the pathologic condition involving the periodontal tissues and the elimination of the underlying factors by postoperative care. Preliminary to any treatment a complete radiographic survey is recommended, to be followed by a thorough prophylactic treatment and a study of occlusion.

Treatment consists in the correction of occlusal abnormalities, subgingival curettement of all involved root surfaces for the purpose of removing all calculary deposits and necrotic material, restoration of proximal contact points and the breaking up of the epithelial lining of the inner wall of the pocket. For this latter operation a sodium sulphide solution is recommended. Surgical treatment of advanced cases is advised, especially in the molar region. Rearrangement of the diet in such a way as to avoid the combination of carbohydrates and proteins is believed to be helpful in these cases. Great stress is laid on the postoperative care, the statement being made that carelessness in personal care inevitably leads to reinfection. The control of the etiologic factors such as bacterial activity and low tissue tone are said to be absolutely essential to the maintenance of health in the periodontal tissues.

Case Report[1]

By Frank Kaiser, D. D. S.
Los Angeles, California

E. B.—This patient presented February 12, 1932, complaining of pain, and brought an X-ray of a devitalized lower left first molar (Fig. 1). She was not in a frame of mind to be dealt with as a diagnostic patient, and consequently, in order to relieve her of her difficulty, it was decided to extract the lower left first molar. At the same time the taking of X-rays of the upper left molar area was suggested, which was done, (Fig. 2), but she refused to have a bacteriologic examination of the periodontal pocket in the upper left molar area, which we suspected as being an acute Vincent's infection and the primary cause of her pain. The patient developed extreme post operative soreness which was the result of a dry socket.

Fig. 1

Fig. 2

Fig. 1. X-ray of lower left first molar, extracted Feb. 20, 1932.
Fig. 2. X-ray of upper left first molar showing large granuloma on disto-buccal root.

History and Clinical Examination:

Age, 25. *Occupation,* School Teacher. *Height,* 5′ 2½″. *Weight,* 122 pounds.

The patient complained of general malaise of four years' standing, which necessitated her sleeping approximately ten hours a day in order to keep going. Her general history was negative, as she had no chronic ailments or organic disturbances of any kind. Her tonsils had been removed seven years prior to this consultation. In 1929 she had a thorough physical examination; the blood test was reported perfect, as was the urinalysis. Four years ago the patient was unhappily married. At the end of a year she had lost eighteen pounds, which was thought to be the result of nervousness; she also complained of a general sore mouth. A dentist at that time treated her for a condition of the gingivae with which he admitted he was not familiar. The patient became discouraged and neglected her mouth, which gradually grew worse. When she presented to me, three years later, the general condition of her teeth was fair, but showed lack of home care. The gingivae presented pockets throughout the mouth particularly in the upper molar areas and in the lower right molar region. She had never been given any

[1]Read before the American Academy of Periodontology, Buffalo, New York, September 9, 1932.

special instructions in regard to home care, but claimed to have used an up-and-down motion approximately two minutes twice a day, which caused her gums to bleed each time. The gingivae were hyperemic with no recession, and there were clinical symptoms of Vincent's.

Roentgenographic Examinations: Revealed a deep socket, including the bifurcation of the upper left first molar, which was also devitalized, but no attempt had been made to fill the root canals. The upper right second bicuspid was devitalized, presenting a distal curvature of the root and a partially filled root canal, which showed a rarefying osteitis of the process at the apex. There was resorption of the crest of the alveolar process in the upper molar and lower right molar areas. All other areas negative.

Bacteriologic Examination (February 19, 1932): Microscopic examination of smear from gingivae showed many pus cells, (Figs. 3 and 4), moderate number of Gram negative and Gram positive cocci, a moderate number of staphylococci, diplococci, and streptococci; many streptobacilli, fusiform bacilli, and Vincent's. spirilla. Diagnosis: Mixed Infection.

Vitality Test: Results of vitality test showed that remaining teeth not previously mentioned as devitalized were vital and normal, with the exception of the lower left third molar and upper left lateral, which were hyperemic, probably due to trauma which was later corrected.

Treatment: Treatment included sub-gingival therapy to reduce the infection, lessons in home care, extraction of the upper left first molar, upper right second bicuspid, and upper right third molar, and a surgical resection on the upper left bicuspid and molar, the upper right bicuspid and molar, and a thorough scaling and polishing of all remaining teeth.

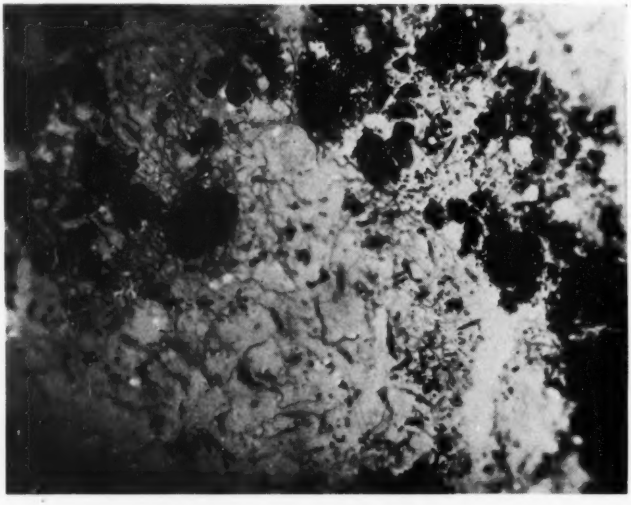


FIG. 3. Gram stain. Pre-operative, pre-medicative slide, Feb. 20, 1932.

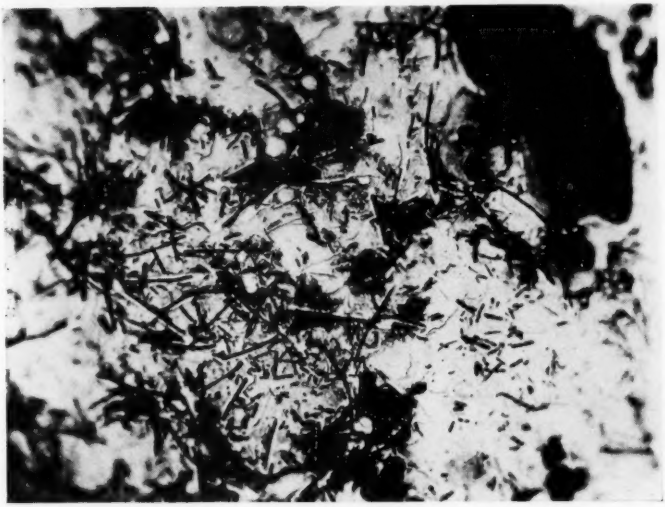


FIG. 4. Carbol-fuchsin stain. Pre-operative, pre-medicative slide, Feb. 20, 1932.

A check-up slide was made on March 23, 1932, (Fig. 5), which was as follows: Microscopic examination of smear from gingivae showed few pus cells, moderate number of staphylococci, of diplococci, and a few fusiform bacilli. Diagnosis: Negative.

On April 22, the occlusion was checked and the patient dismissed. She presented again on June 29, for examination, at which time it was found that she

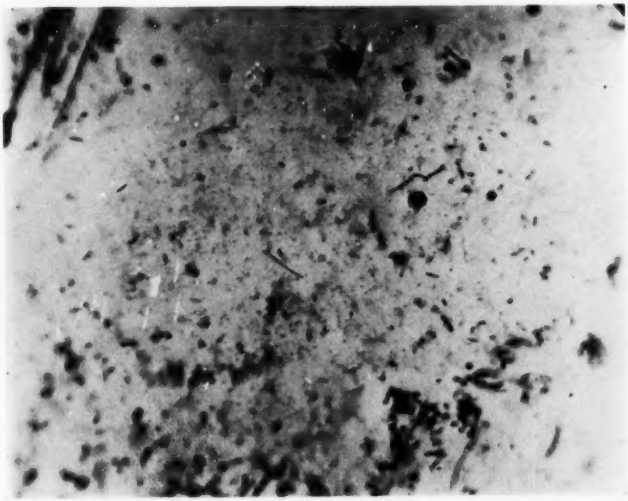


FIG. 5. Carbol-fuchsin stain. Pre-operative, post-medicative slide, March 23, 1932.

had improved considerably, her malaise had entirely disappeared, and her general condition had completely changed. On August 4, 1932, the patient presented for prophylaxis, and a photograph was taken of her teeth, (Fig. 6). Bite-wing X-rays, (Fig. 7a, 7b), and a check-up slide were made on August 22nd, (Fig. 8), which latter showed few pus cells, a moderate number of staphylococci, a moderate number of diplococci, and a few short streptococci. Diagnosis: Negative.

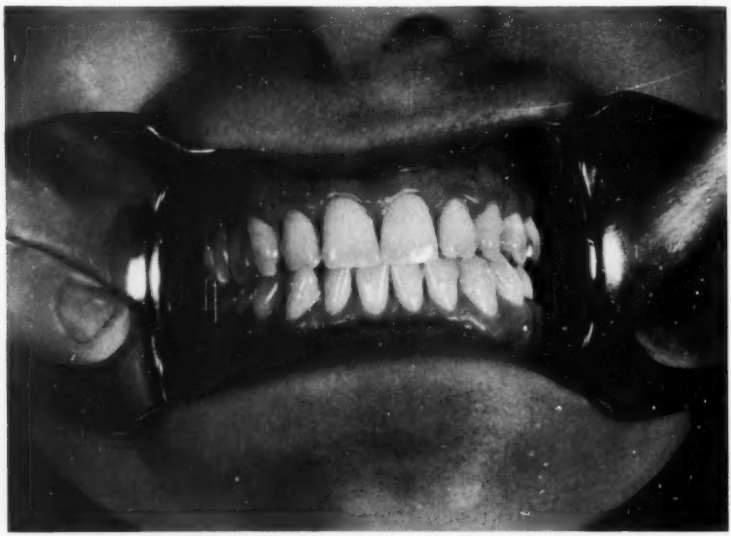
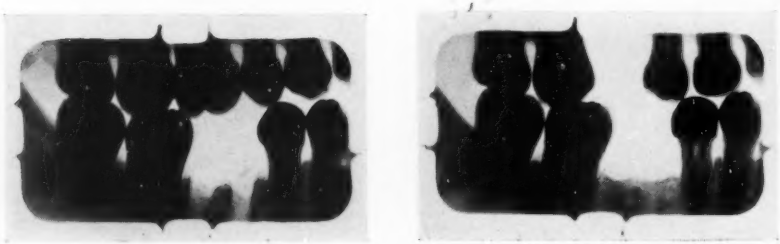


FIG. 6. Post-operative photograph, 5 months after operation, Aug. 4, 1932.



A B

FIG. 7a. Bite-wing X-rays,—left molar—bicuspid area, Feb. 20, 1932.
FIG. 7b. Bite-wing X-rays,—left molar—bicuspid area, Aug. 22, 1932.

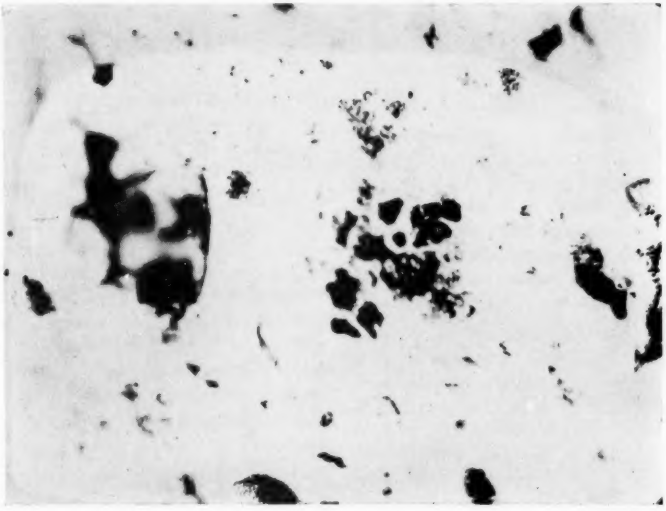


FIGURE 8. Carbol-fuchsin stain. Check-up bacteriological slide, Aug. 22, 1932.

Incidents of Office Practice[1]

Gingival Infection of Unusual Origin

By Arvah S. Hopkins, D. D. S.
Gulfport, Miss.

In presenting this case which I believe to be unique as to its probable causative agent and clinical manifestations, I do not wish to make dogmatic statements to be accepted without examination.

In looking up the literature on Fungus infection of the gingivae, one does not find voluminous reports as in cases of periodontoclasia or Vincent's infection. In fact, I have not been able to find much, and no case with which I could make a fair comparison.

History of the case:

The Cuban schooner Mariana having been disabled had drifted about in the Gulf for some time. The food supply of the sailors had run very low and the hygienic conditions of the boat were bad. After arrival in port, the local ship chandler took the men in charge and noticed that the captain of the boat did not seem to be feeling well. He also observed a peculiar condition of the gums, which alarmed him to such an extent that he gave him a separate tumbler to use while he remained in port. I mention the above to show how conspicuous the condition was. After several days without improvement, the ship chandler sent him to me. When he arrived I saw a man of considerable physical strength of about fifty years of age and after he was seated in the chair, I secured a brief history of a general malaise and a tender condition of the mouth.

His gums appeared to be covered with a roll of cotton about the size of a pencil, both the upper and the lower gums being covered with this peculiar growth.

I had a physician who was doing bacteriological work make a smear to find, if he could, the offending organism. Then I proceeded to clean up this mouth, removing the growth by mechanical means and the spray. At the time I thought I had some type of Vincent's infection to deal with, and proceeded on that theory.

As we all know the gums of a patient suffering with a Vincent's infection in the acute stage are very painful and bleed at the slightest touch. After removing a few of the membranous like growths, I inquired if he suffered any pain. To my surprise he said he suffered very little; this of course was contrary to what I expected. I noticed too that there was very little hemorrhage which is also contrary to an acute Vincent's infection.

The growth did not appear to be firmly fixed to the gingivae, but rested rather lightly with only a small attachment, and when removed did not leave a raw appearance as in cases of acute Vincent's infection. The essential thing to be noticed is that there was very little pain, and only a slight bleeding when the growths were removed with the cotton pliers. The gums were not seriously inflamed.

After removing the growths, (and it required two hours of concentrated effort to do so) I dried the area with cotton rolls, and applied a ten percent solution of potassium permanganate to the gingivae. I still thought I had some form of Vincent's infection. I dismissed the patient until the next day, wondering what the day would bring forth.

On the following day I received the report from the laboratory and found that I was not dealing with a case of Vincent's infection. The bacteriologist reported very few of the spirochetes of Vincent or fusiform bacilli. On the contrary, he found enormous numbers of the organisms belonging to the yeast family, *Schizomycetes* (fission fungi) and *Blastomycetes* (budding fungi).

At the next visit of the patient, I found the growth had been greatly reduced and the mouth had a much more normal appearance than it would have had, had I been treating a Vincent's infection. I removed

[1]Read before the American Academy of Periodontology, Buffalo, N. Y., September 8, 1932.

what growths there were, and again applied potassium permanganate, and dismissed him until the next day.

Most of the false membranes cleared up quickly but there remained several small areas in the upper molar regions on the distal surface that persisted in returning. However after seven thorough treatments of about one hour each his mouth had entirely cleared up, and his general condition improved. There was no swelling of the tissues and no lymph node involvement.

History

In the January Cosmos of 1932, Dr. Lester Cahn of New York discusses fungus infections of the gums. In the cases reported by him the infection seemed to be deeper in the tissues and did not produce the very loose growths as in the above case. Also there was no erosion of the subgingival epithelium as reported in his cases.

In the January Journal of Periodontology volume two, page one hundred sixty-nine, Saravel of Venice, reports a case of fungus stomatitis, which he believes to be rare. In his case, however, he stated that the lesions may be confused with proud flesh which occurs near the roots of carious teeth. However there was no such danger of confusion in this case. He also states that there was no pain. In the case reported in the Dental Cosmos, there was pain and hemorrhage.

In the Journal of the A. D. A., March 8th, 1932, there is a case reported of a serious general infection by Monilia following extraction of a tooth. There seems to be an increasing amount of literature on mouth infections of fungus origin; one should therefore be on the watch for this rare infection.

The Necessity of Psycho-Analyzing One's Patients

BY J. A. JACOBSMEYER, D. D. S.
St. Louis, Missouri

The concensus of opinion is that neither the operative procedure nor the establishment of gums and teeth in a fairly normal condition is our most important work. The imperative thing is the maintenance of a normal mouth by the patient after treatment has been completed. This being true, it is clear, therefore, that the essential duty which confronts us, is to thoroughly imbue patients with an appreciation of the value and necessity of daily mouth hygiene, tooth and gum brushing and stimulation. This implies that we must not only be good operators but good teachers possessed of ability to analyze characters and dispositions, and to recognize idiosyncrasies.

Teaching people to think and to use motor skill correctly is an art. All people do not grasp things with the same facility; neither is it possible to mould the mental processes of all persons with equal ease. To teach successfully, we must capture the attention as well as the favor of the person whom we wish to interest, and then draw that attention to a focus, which in this case is tooth-brushing. Our talk must be fast moving to such a point that their expectation will be aroused for it is human nature to be eager to achieve something new.

To convey information to others, we must understand all types of human minds. We come in contact with people of many different types, viz., the affable, the emotional, the optimistic, the clever, the unreasonable, the cranky, the excitable, the pessimistic, the stupid, the neurotic, the spineless, the self-centered, the self-satisfied, the dissatisfied, the hypocritical, and others. Some are possessed of a high degree of intelligence but a minimum of motor skill. Human nature has many complexes. Among these might be memtioned the temperamental, the hysterical, the undecided, the demanding, the un-coöperative, the morbid, the abusive, the over-enthusiastic, the fearful, and the resistive types. The full of praise patient, the one who continually criticizes those whose services he received previously, the individual with a thousand ailments, the bargain-hunter, the person who knows good dentistry but is unwilling to pay for it, are all met with in daily practice.

As people are not born alike, neither do

they progress through life at the same grade. Again, a person has mental differences at different periods of his life, and even at different hours in the day. The weather, the climate, the atmospheric conditions of the office, ones own temperament, and many other factors affect the patient. It is our duty, therefore, to understand various temperaments and handle patients accordingly. Again, we must learn to sense whether a patient is in a receptive mood at the particular time he is in the chair. In a word, we must be possessed of a versatile temperament to be able to control if possible all types of patients.

The human machine, like any piece of machinery, responds in characteristic ways to forces that act upon it. What its responses will be to any situation can be determined in a general way by a study of the following four factors:

1. The kind of machine;
2. The things with which the machine comes in contact;
3. The chemical changes through which it passes;
4. The immediate elements which constitute the situation.

In other words, we must have an understanding of ourselves and our patients. In regard to the latter, we must realize that our first duty to them is to have an office with a bright and cheerful atmosphere; we owe it to them also to come to work mentally and physically in shape to meet with any and all conditions which may arise in connection with the day's routine. We have all had days when we entered our offices mentally fatigued; fagged to such a point that we dreaded the day's activities, and everything we did appeared in the light of drudgery. This frame of mind is interpreted as an anxiety neurosis, and this mental attitude accompanied by its characteristic facial expression, and consequent behavior, exerts an effect on patients. They develop an anxiety complex, become fearful, reluctant, restless, sometimes even inattentive.

We must realize that many patients enter our office a bit timid and fearful. To offset their condition, we must radiate twice the ordinary amount of mental strength, for unquestionably cheerfulness tends to make patients more self-assured and comfortable. Realizing this, it is easy to see what a drain there is at all times on our physical and mental resources. If one is mentally charged, his work cannot be a drudgery as he can without difficulty overcome the disagreeable features of his work, as well as overcome the phobias displayed consciously or unconsciously by his patients. Of course, it is of the utmost importance to avoid all clashes of temperament.

Now for a brief discussion on our contact with patients. Patients like to have the floor. They are eager to unfold their troubles, and in our response, we should attempt to establish a feeling of confidence in the work which we propose to do. This can best be done by our saying little—that is by being a good listener. A sitting cannot be completed successfully with a distressed, moody person. When such types enter our offices, it is well to engage in light conversation so as to make them happy and cheerful. Failing in this, terminate the visit as quickly as possible and have the patient return at a later date. Instilling hope and creating an incentive or want for the lesson at hand is the first step, but remember, our appearance and disposition have a tremendous effect on the patient. If sufficient effort is made, almost every patient can be carried into the mood desired, but this implies having entire command of the situation. Strength of mind and an understanding of others is essential if we are to keep things in hand and avoid the danger of either rudeness or boredom.

Having produced in the patient an eagerness for information, we must employ the most effective way of imparting it. We must establish the correct mental picture and instill the lesson deeply enough so the patient will continue its application when not under our supervision. To tell people to brush their teeth frequently will not turn the trick—the problem is more difficult than that. Patients must be influenced to the point that they are changed from irresponsible to responsible beings regarding mouth hygiene. This means they must be induced to alter their accustomed, ingrained manner of living in regard to health habits. It is well for us to remember there is an opening wedge for each person, which if

found, will enable us to impart our lesson successfully.

We are all creatures of habit—in fact, we may be called a complex system of habits. Therefore, to make a change in anyone, we must get at his habit system. To awaken a patient to the necessity of frequent and regular tooth and gum brushing, we must provide for him a stimulus; then we will get a response. This may be done by starting with his present condition, next taking up something which will make a difference in that condition, and finally touching on an effect which presupposes a cause, or a cause which must necessarily be followed by an effect. By this time, we can judge whether the patient is interested or not. This is known as carrying them over the dead line, and if we have been successful in our efforts, we have awakened an eagerness for information.

Give a human being a lot of facts regarding his teeth and health, and while he may be quite indifferent you will find that when shown a perfect mouth, with emphasis placed on its being an aid to beauty, physical fitness or even social success, the patient will literally lap up the information given him. While scolding makes you feel comfortably superior, I question if it ever really accomplishes any purpose, other than to put the other person on the defensive. On the other hand, praise given is often far more effective, for it is true that we all derive pleasure from the attitude which our fellow-beings have toward us. The desire to be well thought of by our associates may be based on pride, but it is fundamental, and it certainly does serve as a stimulus to keep a patient up to the mark.

Usually a successful contact with patients can be made by the following steps:

1. Make the person to whom you are talking, feel your genuine interest in him by giving him the impression that your time is his entirely for as long as he wishes.
2. Establish some bond of common interest or experience.
3. Find something about the person's looks, manner or experience which you can conscientiously admire.
4. Try to make your patient feel your desire to help him.
5. Make him realize you believe in him and above all, make him believe in himself.

Having established a point of contact with the patient, we must analyze to him what we wish to accomplish and the logical reasons for the new habit, and then explain the procedure to be followed. In this connection it is important to remember that only a small amount of information can be given at one sitting, as over-crowding the mind defeats our purpose. At the same time it is necessary to over-stress the important points so as to offset the information which will be forgotten. If one is trying to teach toothbrushing, for instance, only a few points of the technique should be given at one time so these will be firmly imbedded in the mind. My experience thus far has shown me that five visits are necessary in order to really impart the lesson.

For this reason it has seemed to me advisable to set one room aside for the exclusive teaching of toothbrushing as this is such an important phase of our work. In this room is only such equipment as is necessary. Most people are visual-minded for anything presented to the mind through the eye has a far greater chance of being retained or recalled than that presented through the other senses. In pursuance of this knowledge, we realize that pictures are the simplest and clearest means of transmitting ideas. It has been said that "A picture, with a few words of explanation, will make it possible to get over an idea in one minute that would require two minutes without the picture".

In order that a picture stimulate interest or arouse attention, it must be given full opportunity to accomplish its purpose. In this connection it is well to follow the plan of an art director in a well-known women's college. Rather than crowd the gallery wall, she has a room set apart, and in that room one picture is hung—usually for an entire week. In front of that picture, at a great enough distance, is a bench with a back. The director knows that if a new and rare experience is to be incorporated, we must be given time, and that if we take

time, the rest of our bodily organism must not be crying out for attention.

If one is trying to teach toothbrushing, it is well in addition to pictures, to present a perfect model of motor skill in this line. Teach the patient to criticise his own method and the results which he achieves. Emphasize the length of time necessary for thorough tooth and gum brushing, warning the patient against trying to do the operation too speedily. To make this a daily life habit, we must encourage, and in some instances, praise the patient so that he will reach the desired goal. Failing by either of these methods to secure the response we seek, we may find it necessary to use the shock plan.

In the brief time allotted for this discussion, it is impossible for me to attempt to analyze all the various kinds of temperament, but I believe the man who has the ability to recognize the different types has the ability to cope successfully with them.

Case Reports of Nutritional Deficiency as Related to Periodontia

BY JOHN B. WEST, D. D. S.
Elmira, New York

The research work of McCollum, McCarrison, Howe, Mellanby, Drain, Price, Davis, and others has convinced me that the principles of nutrition if applied to the problems of dentistry will materially assist in their solution.

For several years, as a clinician, I have carried out in my practice and in my personal dietary the principles of nutrition presented in these case reports. From these clinical observations I am convinced of the reasonableness of this line of attack not only in controling incipient periodontal lesions but dental caries also.

CASE REPORTS

Case 1. Girl, age 5 years. Developed a decided limp in right sacro-iliac joint. Orthopedist in neighboring city had roentgenograms taken and made a diagnosis of tuberculosis of sacro-iliac joint and advised placing the hip in a plaster cast for at least one year. This advice the parents hesitated to follow, and consulted me for advice. Mouth examination revealed the deciduous teeth completely decayed off at the gingival margins of the gum with dark colored roots exposed. The color of the mucous membranes indicated anemia, the etiology of which, I judged to be nutritional. The dietary history revealed, that although the child lived on a farm, she did not drink milk as she did not like it. She did not use green vegetables or fruits. Her diet consisted of carbo-hydrates; white bread, cookies, potatoes, preserves, desserts, cake, and candy. From this history I decided that the child was deficient in iron and calcium together with the vitamines A, C and D. Treatment advised was an 8 oz. glass of orange juice per day; at least three 8 oz. glasses of raw milk taken from one cow, which was fed so as to stock her milk with the maximum amount of minerals and vitamines. This was supplemented with small amounts of meat, leafy green vegetables, tubers, and whole wheat bread; no candy or pastries. Two teaspoonfuls of cod liver oil activated in sunlight was prescribed per day. A sun suit was put on the child and beginning with a very short time in the sunlight this was gradually increased. Recovery of the sacro-iliac joint has been complete without the use of a plaster cast and the permanent dentition has erupted in a satisfactory manner. The child is now 14 years of age, nine years having elapsed since beginning treatment; enough time to prove the efficaciousness of the treatment.

Case 2. Female, single; age 23; student; Complaint: spongy and bleeding gums. Mouth examination revealed a full dentition, with not an excessive amount of dental caries. Mucous membranes anemic, gums swollen at the gingivae and bled easily when they were touched.

History: Constipation. Slight rheumatic pain in left arm. Frontal headaches constant. Had had several attacks of indigestion. Very nervous. Painful menstrual periods. Diet consisted of dry toast, white bread, chocolate, cake, pie, and some meat.

General examination: Tenderness along

the lower border of the liver and over the ovarian regions. Temperature 97 4/5°. Blood pressure, 108 systolic. Haemoglobin 80%. Erythrocytes 5,100,000. Leukocytes 6,000.

Urinalysis: Sp. Gr. 1.008; Degree of Acidity 36. Albumen-trace. Urea .5 of 1%.

Treatment: First a thorough oral prophylaxis. Dietary treatment consisted of putting the patient on one quart of grade A raw milk per day, plenty of fresh fruits especially oranges, June butter, whole wheat bread, leafy green vegetables; no white bread or potatoes. Two No. 00 capsules of activated cod liver oil twice daily after meals and 10 grains of a compound of calcium and phosphorus once a day before meals.

Progress: Within three weeks gums stopped bleeding, nervousness decreased, and frontal headaches cleared up. Constipation lessened under the use of two quart warm saline enemas daily on retiring. On the whole, patient's condition in every particular improved.

Case 3. Female: Married; age 34. Complaint: Persistent pain spreading over the left side of face.

History: Patient had been treated by several physicians and dentists without any permanent relief. Upper left third molar had been extracted as an etiological factor but pain had increased since the extraction.

Mouth examination: The lower left first molar had been extracted with a resulting migration of the lower second molar and a malocclusion of the upper left second bicuspid and the upper left first and second molars. Teeth showed considerable tendency to caries.

General history: Persistent chronic constipation. Rheumatic pains in various parts of the body. Very nervous. Nauseated feeling a great deal of the time. Painful menstrual periods. Diet had consisted of meat, potatoes, fruit, with some pastries and sweets; no milk.

General examination: Temperature normal. Blood pressure 94 mm systolic. Blood count, erythrocytes 7,500,000, leucocytes 9,000. Increased second sound of the heart at tricuspid valve in aortic area. Urinalysis: Specific gravity 1.020; Color, slightly cloudy; Reaction, neutral; Indican, Urea, .9 of 1%; no sugar; no albumen.

Diagnosis: Traumatic occlusion as the exciting cause with a predisposing causative factor of a long standing imbalance of calcium.

Treatment: Correction of the traumatic occlusion. Change of diet to a corrective one, including one quart of raw milk per day, as many oranges per day as the patient desired, green leafy raw vegetables, two salads per day, baked potato or whole wheat bread if the patient desired, and meat not more than once per day consisting of either lamb or chicken. Ten grains of a combination of calcium and phosphorus per day were prescribed, before meals, and two No. 00 capsules of activated cod liver oil after meals.

Progress: After four days the pain in the left side of the face was spasmodic, a great deal of the time entirely gone. At the end of ten days it disappeared and has not returned. At the end of six weeks the patient had so much improved as to make her feel like a different person. Painful menstrual periods had disappeared and patient reported that her health had not been so good in years.

Case 4. Female; married; age 28. Complaint: Spongy and bleeding gums. Mouth examination: Teeth looked well cared for with very little calculus formation; occlusion normal; very few fillings. General examination: Temperature normal. Blood pressure: 112 mm systolic, 70 mm diastolic. Hemoglobin 90%. Blood count: erythrocytes 4,200,000; Leucocytes 7.600; Urinalysis: Specific Gravity 1.919; Color, very turbid due to phosphates. Reaction, acid. Degree of acidity 50; Negative as to sugar, indican, and albumen. Patient complained of a great deal of headache, chronic constipation, biliousness, and distress in the stomach. Very nervous. Tenderness over the ovarian region, over the region of the appendix, and along the lower border of the liver.

This patient's diet had consisted as follows: Breakfast: bacon, eggs, rolls, and coffee. Dinner: meat, cooked vegetables and white bread; no dessert. Supper: meat, vegetables, sometimes salad, and rich des-

serts. The patient took no exercise, weight 205 lbs.

Treatment: A thorough oral prophylactic treatment. Changed diet to a corrective one. Six glasses of water prescribed per day. At first no breakfast but a pint of hot water with a tablespoonful of sodium bi-carbonate, this to be followed by several glasses of hot water. If after drinking the water the bowels did not move in a half-hour a luke warm enema, two quarts of water to one tablespoonful of sodium bi-carbonate was prescribed. This enema was also advised on retiring. About eleven o'clock a glass of orange juice. This treatment was given to help alkalize the body.

The mid-day meal was to consist of baked potatoes, green leafy vegetables, raw salads, beets, carrots, fresh fruits; no liquids. Dinner: small piece of lamb, green leaves of lettuce, sauerkraut salad, no bread or starches, and one pat of butter. Systematic exercise was prescribed.

Progress: Within three weeks all bleeding and spongy conditions of the gums had cleared up. The patient gradually lost weight and in six months weighed 165 lbs. All tenderness over the areas mentioned cleared up. The bowel movements approached the normal. Headaches and biliousness disappeared. Color of the skin lost its muddy appearance and assumed a normal healthy one.

I am well aware that the report of only four cases means very little in the establishment of the reasonableness of this line of treatment of periodontal conditions; but these cases are taken from a group of a considerable number that have convinced me that the results attained are not the result of accident or co-incident.

Periodontal Foci of Infection

BY SAMUEL C. MILLER, D. D. S.
New York City

Although focal infection from the apices of teeth has been looked upon as an important phase of pathology for many years, the disastrous systemic disturbances which might be produced due to the absorption of bacteria and their toxins from periodontal pockets have been given comparatively slight attention.

At the New York University Dental Clinic and at the office, I have found numerous cases which have been extremely benefited either by periodontal treatment which eradicated the pus pockets or by extraction of teeth so involved, when the prognosis warranted.

Bacteriologic examination made from the pockets around teeth so involved has revealed a large variety of organisms, chief among which were those of the staphylococcus and streptococcus groups. Mixed vaccines made from these cases gave no local benefit, but produced results solely in relieving the focal infection diseases, and this only after the eradication of the primary focus. No beneficial results were obtained in my hands, in the alleviation of the periodontal disturbance itself through either autogenous or stock vaccines, although several writers, both here and abroad, have claimed success with this means of therapy. We at the school, have always felt that, although the infection was present, it was only there because of the lowered tissue resistance produced by one or more primary etiologic factors, and would be eradicated when these factors were removed and tissue health re-established. Proceeding on this assumption, success has been obtained in the alleviation of rheumatism, neuritis, inflammatory arthritis and other systemic conditions which were found to be due to periodontal foci, by the institution of periodontal treatment alone, without the use of any antiseptics or vaccines.

The following case is a striking example of the importance of the removal of periodontal foci:

Patient: Male; White; Married; Age 35; Occupation: chauffeur; appeared at the New York University Dental Clinic for diagnosis. He had been under the care of a physician for *spondylitis deformans*,

(deforming arthritis of the spine). His deformity was so severe that it had been impossible for him to work during the previous six months. This, together with the drain which medical attention had put upon his meagre purse, had brought this man, who was married and the father of two children, to a state of extreme poverty.

Clinical dental examination revealed a generalized superficial periodontal disturbance with no pocket more than three millimeters deep. Radiographic examination revealed some periapical thickenings but, since all the teeth gave normal responses for vitality, no immediate extractions were advised.

The case was put in the hands of a Senior student, who concentrated upon the periodontic treatment, seeing the patient practically every day. With much gratification to all concerned, there was a rapid and continual improvement in the spinal condition until, in two weeks, the patient was able to walk erect with perfect comfort. At this time he reported that he had been reinstated in his old position and had already resumed his work. Recent reports show no recurrence.

Opportunity Neglected

By H. H. Cleaveland, D. D. S.
Springfield, Mass.

Nothing is presented in this paper which requires very deep thinking. It is not a contribution to scientific achievement. I wish only to call your attention to a neglected opportunity which has about the same relative value in the practice of our profession that a small puncture below the water line of a canvas canoe has when voyaging far from land on some great northern lake.

Differ from me if you will, but it is my belief that many of us strive most conscientiously to develop in the mind of each patient the idea that the professional part of our service is more valuable than our craftsmanship, and then with a thoughtlessness that is colossal, we destroy the beautiful creation before the patient leaves the chair.

Dentistry can advance only as rapidly as the public appreciates the service rendered, but even where the periodontist has been most fortunate in the surgical treatment of some particular case of periodontoclasia the patient generally believes the disappearance of the disease is due to improved general health or the use of some proprietary preparation, such as an antiseptic mouth wash or tooth paste.

Several years ago an elderly woman with well developed periodontoclasia appeared with the request that the mouth be made as healthful as possible, as she was leaving at the end of four days to make her home in a distant city. After several hours work during the four days, some discomfort might be expected, though she made no complaint. I gave her a 2 oz. bottle of highly flavored and colored zinc solution to use while traveling, but being anxious to know the result of my work, I asked her to write me as to the health of her mouth in about three months. A most conscientious woman, she remembered and wrote, "Dear Doctor: The little bottle of medicine completely cured my mouth."

For another patient, the necessary treatment was completed. Thorough and frequent use of toothbrush and dental floss was persistently taught, and because of a condition in which soda in solution might be helpful, it was recommended and the patient discharged.

Some three months later she appeared and reported: "My gums are in perfect health and my teeth are no longer loose. I think the soda solution is perfectly marvelous and I am trying to get all my friends to use it."

Certainly these were not usual cases, but they illustrate weak links in a chain which should have no weakness of which we are conscious.

If there is improvement following surgical treatment or if there is obvious improvement after the application of a scientific technique of brush usage, the credit for

increased health must be kept where it belongs, if the periodontist is to receive any special recognition from the patients whom he serves.

I wonder if the promiscuous recommendation of mouth washes and tooth pastes by the general practitioner of dentistry, and even by the periodontist may not be a weak link in our education of the public.

These preparations might be described as one author described a character in his book; "She was a woman of great though unavailing virtues and small but unbearable sins."

Have you ever had this experience? After having advised your patient as to diet, exercise, rest, moderation in the use of alcohol and tobacco, or even the total abstinence; demonstrated how to use the floss effectively and without danger to the soft tissues; emphasized the frequency and thoroughness with which the brush should be used to cleanse and to massage;—and then did this patient look up at you with a soulful expression and say, "No one ever explained to me so clearly and convincingly as you have done and it all sounds so logical that I want to follow your instructions in every way. What mouth wash or tooth paste do you recommend?"

We have heard much about increased education of the dental student, the dentist and the laiety. It seems barely possible that there is still a field for further effort in the attempt to *eliminate erroneous information*, or belief, which is my reason and at the same time my apology for this paper.

In Memoriam

SIMON ESCHELMAN, M. D., D. D. S.

It is with profound regret that we note the passing of an Honorary member of the American Academy of Periodontology—Dr. Simon Eschelman of Buffalo, New York, on September 26, 1932 at the age of eighty years. Dr. Eschelman was a graduate of Temple University school of dentistry, the Royal College of Dental Surgeons, Toronto, and the University of Vermont School of Medicine. He was one of the earliest members of the American Academy of Periodontology and a charter member of the American College of Dentists. He was a past-president of the Buffalo and Eight District Dental Societies of the State of New York. Mrs. Eschelman and two sons, Karl F., D. D. S., M. D. and Leo S., D. D. S., survive.

BOOK REVIEWS

Oral Spirochetes and Related Organisms in Fuso-Spirochetal Disease, By David T. Smith, M. D. Associate Professor of Medicine, Duke University-Medical School, Durham, N. C. Formerly Bacteriologist and Pathologist to the New York State Hospital for Incipient Tuberculosis, Ray Brook, N. Y. 1932.

The material in this pioneer work on Oral Spirochetosis by an American author was collected and prepared largely while Dr. Smith was on the staff of the New York State Hospital for Incipient Tuberculosis making possible a splendid combination of laboratory and clinical material by which he could study cases from all angles. The result is a painstaking, comprehensive book.

The main points concerning diagnosis, treatment and prevention may be summed up thus:—

1. The diagnosis is easily established by microscopic examination after staining, or with the darkfield.

2. The treatment is fundamentally the same in all the various types of lesions although necessarily modified by the structure and location of the organ involved. Open drainage and free access of air is indicated; supplemented by local application of oxidizing agents and by arsenic therapy.

3. This is a preventible disease.

The author states that, "Oral Spirochetes alone do not produce disease in man; but they are probably the most important member in a symbiotic group of anërobic organisms which is capable of initiating a severe and often fatal disease in man."

The extent of the invasion of the body by these organisms is indicated by the fact that there are thirty-two chapter titles embracing almost as many organs and tissues, among the most important of which are the oral tissues.

Paradentosenbehandlung mit Besonderer Berücksichtung der Entlastung. Treatment of Paradentoses with Especial Consideration to Relief of Trauma. Kiefer, E., Director of the Zahnärztlichen Fortbildungs-Institutes des Reichsverbandes der Zahnärzte Deutschlands. Berlin. 110 pp.; 65 illustrations. 1932.

Anyone who concerns himself with the treatment of Periodontal diseases will greet with pleasure and satisfaction this recently published manual in which the author gives proper emphasis to the relief of trauma as a therapeutic measure applicable to these conditions. "The etiologic impulses which determine the pathologic picture of paradentosis are not yet entirely clear but the general opinion is that endogenous and local factors must combine to produce a paradentosis. The therapy must therefore attempt to dispose of the endogenous background, and to eliminate the causative factors and symptoms which lie in the mouth itself. A systematically carried out paradentosis treatment then should be divided into three parts—Internal treatment, Relief of strain and Local treatment." Until recently the most particular attention was given to the elimination of pockets, while trauma relief was rather neglected. The ideas concerning correction of occlusion were much too narrow and the same may be said of the types of stabilizing and relieving *splints* (among which the author includes corrective bridges and other appliances.) These *splints* constitute only one of many means for relief of which the object in every case is the restoration or the production of a balanced function-occlusion. Here one is strikingly impressed with the necessity for the periodontist's understanding of at least the elements of orthodontia. The author has assembled a series of casts of typical cases such as one finds regularly in practice and various methods of bringing about balanced occlusion are demonstrated. All possible methods of local treatment of these cases are described (deep scaling, curettage, Dunlop's, gingivectomy, etc.) but

without evaluation of their respective merits.

Dr. Kiefer has full command of his subject and the manual is unusually clear and will be found most useful to the practitioner.

Histopathology of the Teeth and Their Surrounding Structures; 479 pp; 385 engravings. 1933.

Rudolf Kronfeld, M. D., Prof. of Special Histopathology, Director of Department of Research, Chicago College of Dental Surgery, Chicago, Ill.; former Assistant at the Dental Institute of the University of Vienna, Austria.

Dr. Kronfeld's work is already well and favorably known through his contributions to our journals and the present volume is a most interesting and valuable addition to the growing list of books by the men of Gottlieb's Vienna school. A comprehensive review of this book will appear in the next issue of the Journal of Periodontology.

Diseases of the Mouth, Fourth Edition. By Sterling V. Mead, D. D. S., M. S., B. S. Professor of Oral Surgery, Georgetown University Dental School. 1932. pp. 932 with 523 illustrations and 40 colour plates.

This edition of Mead's Diseases of the Mouth is the fourth to appear since 1927 which in itself bears testimony to the excellence of the work. The text has been revised, elaborated and enlarged until the present volume is almost twice the size of the original with correspondingly more illustrations and colour plates. Though primarily intended as a text-book, for students of dentistry and medicine, it will be found invaluable for reference in practice. In his preface the author states that he had in mind the growing need for better coöperation between dentist and physician and this object is kept constantly in view. Thus the opening chapter on Examination includes urinalysis, and blood and serological tests in addition to the history and thorough charting of all phases of the oral condition —mucous membranes, tongue, saliva, general condition of teeth, type of occlusion, shape and condition of arches and other data. Then follow chapters detailing the making of X-rays, pulp tests, trans-illumination and bacteriologic examination; all of which must be brought together and coördinated before intelligent diagnosis can be made. Of the X-ray, Mead wisely says that it is valuable in oral diagnosis only when interpreted by one thoroughly schooled in dentistry and who understands the technic as well as what the X-ray really shows, and knows its defects and limitations. He lays great stress on transillumination as a diagnostic aid and cites and illustrates many pathologic conditions which were disclosed by this method where X-rays were negative. An interesting suggestion is the use of transillumination to follow up and observe gradual change to normal translucency of congested areas in the soft tissues. As a diagnostic aid Mead says, "Transillumination far excels radiography in the early stages of periodontic infection."

There is a brief practical chapter on Bacteriology.

Specific infectious diseases are given under a special heading and the part devoted to syphilis is noteworthy for its minute and accurate descriptions and illustrations of the different oral manifestations of the disease.

The chapter on Periodontal Diseases is but slightly changed from the earlier edition beyond the addition of new illustrations. The subject is admirably handled and while Mead's arrangement and classification is not orthodox, it is logical, scientific, and entirely in keeping with modern teaching. In outlining treatment he is brief and strictly to the point. It is notable that Mead who is essentially a surgeon, recommends radical surgery here only as a last resort. His same conservative attitude is also manifest in the handling of impacted teeth, focal infections and acute infective processes.

The long section on Stomatitis will be found most worth while by the periodontist. It would be difficult to find a more comprehensive treatise and while many conditions given and illustrated are rare, the importance of including them is obvious. The author's general plan of Definition, Etiology, Symptomatology, Diagnosis, Prognosis, and Treatment is followed out for all important conditions, briefly for the most part but to the point. He has improved

and elaborated his presentation of Ulceromembranous Gingivitis to include its bacteriology and incidence, the predisposing effects of diet, etc. He has added material on Streptococcic Gingivitis which he treats as a distinct, often serious pathologic condition, its diagnosis complicated by the fact that it is frequently super-imposed on other lesions. Various drug and mineral poisons and the oral picture of acute eruptive diseases and others are included.

The final chapter on Oral Sepsis and Systemic Disturbances again shows the broad viewpoint of the author. In focal infection cases in particular there is need for coöperation between the dental and medical professions. The physician should not "overlook the value and the advantages of Nature's own teeth, and should remember that no artificial restoration can really equal the tooth which has been extracted." While emphatic in recommending elimination of all infections, Mead does not consider extraction always indicated as the only means of accomplishing this even in the case of the pulpless tooth. He thinks enclosed periapical infections are more dangerous than freely draining periodontal areas in which the oral and gastric secretions in a measure destroy or overcome the virulent effects of the organisms; except where there is continued irritation from excess amount of toxins, or a lowered resistance, in which cases a slight local area of infection may be sufficient to cause general disturbance.

An idea of the vast scope of Mead's work may be had from some of the chapter headings—Diseases of the Blood and Blood Vessels, Diseases of the Nerves, Diseases of the Lip, of the Tongue, of the Throat, of the Mandibular Joint and the Muscles of Mastication, Diseases of the Pulp, Periapical Diseases, Dentition, Tumors and Cysts.

The arrangement of the book is excellent throughout and as a textbook, easy to follow. The author has taken each tissue in turn with all the diseases which are manifested in it and while this necessarily entails some repetition it makes it the more useful as a reference book. The free use of illustrations, many of them in colour, adds measurably to its value, as does the long list of references at the close of each chapter.

Celia Rich, D. D. S.

Human Odontography and Histology. A Text-book for Undergraduates and Graduates in Dentistry and Dental Hygiene. Churchill, Herman R., Tandarts (Utrecht), D. D. S., Dr. Med. Dent. (Rostock). Ass't Prof. of Dental Histology, Histopathology and Comparative Odontology, The Thomas W. Evans Museum and Dental Institute, School of Dentistry, University of Pennsylvania; Oral Pathologist at the American Oncologic Hospital of Philadelphia. 298 pp. 347 ill. 1932.

"The purpose of this book is to give to dental students that knowledge of microscopic and macroscopic anatomy of the human teeth that is indispensable to the solution of their varied dental problems." Contents are as follows:

PART I—Fundamental Principles—The Permanent Teeth—The Deciduous Teeth—The Identification of Specimen Teeth—Occlusion—The Blood, Nerve and Lymph Supply of the Teeth—Appendix.

PART II—Fundamentals of Histology and Embryology—The Dental Structures and Their Means of Support—The Mucous Membranes of the Mouth—The Salivary Glands.

Chemistry of Food and Nutrition. By Henry C. Sherman, Ph. D., Sc. D., Mitchill Professor of Chemistry, Columbia University. Fourth edition. pp. 614, with 32 illustrations. 1932.

This practically rewritten edition, presenting the principles and scientific foundation of the chemistry of foods and nutrition, deserves an enthusiastic welcome. The subject is concisely discussed in a simple manner that conveys a good general understanding of this field. Food is considered chiefly in its relation to nutrition; detailed description of individual foods is omitted to advantage. The food requirements of man and considerations essential to development of good judgment on the nutritive value of foods and their choice and use for the maintenance and advancement of health are prominent features of the plan of presentation. The text is excellently balanced in its treatment of the four main factors of nutrition, energy, protein, mineral elements and vitamins. New material and data and several new chapters

are incorporated. Valuable reference lists for supplementary reading accompany the chapters. Tables on the edible organic nutrients, ash constituents and vitamin content of individual foods are appended. This edition excels the past excellent editions in organization and presentation of subject matter and is an epitome of best opinion and judgment on the science of nutrition and general chemistry of foods. The writer is a master in his field. All directly or indirectly interested in foods and nutrition will want to study this book from cover to cover.

Journal American Medical Association, Vol XCIX: (16)

The Wisdom of the Body. By Walter B. Cannon, M. D., LL. D., George Higginson Professor of Physiology, Harvard Medical School. pp. 312 with 41 illustrations. 1932.

Dr. Cannon's views on homeostasis have been repeatedly considered in the editorial columns of The Journal. There are within the human body numerous stabilizing devices tending to maintain and to restore normality. There is an automatic mechanism for maintaining a constant supply of water, salt, sugar, protein and fat. There are, moreover, factors of safety and of defense, as well as mechanisms for repair. Most of the chapters of Dr. Cannon's book concern these mechanisms as they are involved in relationship to specific substances. His concluding chapter indicates similar forces in biology and in social life. The living human organism suggests that stability is of prime importance. Thus it teaches a lesson to the social organism. The volume represents a sound and philosophical summary of human physiology.

Journal American Medical Association, Vol. XCIX (11)

Man and Medicine: An Introduction to Medical Knowledge. By Dr. Henry E. Sigerist, Professor at the University of Leipzig. Introduction by Dr. William H. Welch, Professor of the History of Medicine, Johns Hopkins University. Translated by Margaret Galt Boise. pp 340. 1932.

Professor Sigerist has just taken the chair of History of medicine in Johns Hopkins University. The book here presented has had a remarkable success in Germany, where there would appear to be a far greater number of intelligent laymen interested in medical knowledge than in this country. The book is divided roughly into parts dealing with man, disease, medical aid and the physician. It presents not only an outline history of medicine, written in exceptionally good English, but also a philosophy of medicine and a vast amount of fundamental medical knowledge. It is hard to believe that any person could be considered well educated without the information that Professor Sigerist here makes available. The layman who wishes a better understanding of current trends in medicine will find in this book the orientation that he desires.

Journal American Medical Association, Vol. XCIX (12)

Food and Character. By Louis Berman, M. D. pp. 357. 1932.

All dentists who have been students of Diet and Nutrition will find much of interest in this volume as the following chapter headings prove: III, The Metabolism of Leadership; VII, Lime and the Neurotic Constitution; VIII, Sugar and the Energetic Personality; IX, Fat, Acidosis and the Phlegmatic Character; XIV, Human Chemistry and Human Destiny.

MEDICAL DICTIONARY

THE AMERICAN ILLUSTRATED MEDICAL DICTIONARY. A Complete Dictionary of the Terms Used in Medicine, Surgery, Dentistry, Pharmacy, Chemistry, Nursing, Veterinary Science, Biology, Medical Biography, Etc., With the Pronunciation, Derivation, and Definition. *By W. A. Newman Dorland, A.M., M.D., F.A.C.S., Lieut.-Colonel, M.R.C., U. S. Army; Member of the Committee on Nomenclature and Classification of Diseases of the American Medical Association; Editor of "American Pocket Medical Dictionary." Sixteenth Edition, Revised and Enlarged with* 941 *Illustrations, In-*

cluding 279 portraits. With the Collaboration of E. C. L. Miller, M.D., Medical College of Virginia. Philadelphia and London.

The Sixteenth revised and enlarged edition of Dorland's *American Illustrated Medical Dictionary* contains more than 3,000 new words, including all the latest technical terms in medical, and dental science and practice.

The terminology conforms to the standards of the American Medical Association and other recognized scientific bodies.

This dictionary, stands foremost in its class and justifies its title as the standard American medical dictionary. As an up-to-the minute book it should find a place in every progressive physician's and dentist's library.

Announcement

THE ANNUAL meeting of the American Academy of Periodontology will be held at the Hotel Stevens, Chicago, August 3-5. Among those whose names appear on the program are Drs. Thomas J. Cook, Philadelphia; W. M. Reppeto, Dallas; Paul R. Stillman, Sidney Sorrin, New York; Frederick W. Merrifield, Milton T. Hanke, Isaac Schcur, Chicago; Willa Yeretsky, Grand Rapids; James A. Sinclair, Ashville; Jesse L. Williams, Jacksonville; Edward L. Ball, Sidney J. Rauh, Cincinnati; A. W. Bryan, Iowa City.

Research Reports, Incidents of Office Practice, Round Table and Topic Discussions as usual. No evening sessions will be held. The Banquet will be Friday evening August 4th.

A cordial invitation is extended to all members of the American Dental Association to attend the scientific sessions of this three-day meeting.

BENJAMIN TISHLER,
President
CLAYTON H. GRACEY,
Secretary

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The July 1933 issue of the Journal of Periodontology will contain 1931 and 1932 abstracts on periodontia.